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THE
LARYNGOSCOPE.

VOL. LVI DECEMBER, 1946. No. 12

NUTRITION IN OPHTHALMOLOGY AND OTOLARYNGOLOGY.*

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The enemies of health are those from without and those from within. Of the former we have abundant knowledge; of the latter — far too little.

In meeting eye, ear, nose and throat problems caused by infection superb work has been done by the bacteriologist, the pathologist, the clinician and the surgeon. This has resulted in a degree of satisfaction with the present teachings and medical and surgical treatment which is perhaps justified. It would be foolish to anticipate no further advances; "Romance is dead, the Cave Man said"—and he was wrong. However, it requires considerable imagination to figure out where to expect marked improvement over present procedures. The only criticism now generally made is that surgery has often proved to be too radical. Furthermore, in many conditions, chemotherapy has proven a great boon — lessening the extent and frequency of surgery. It would be difficult to estimate how far surgical procedure has actually been forestalled by arresting bacterial activity with such drugs as the sulfonamides and penicillin. In otolaryngology, with the ex-

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ceptions of corrective procedures such as septal resection and drainage of pus-containing cavities, surgery now is considered necessary much less often than formerly. In brief, the enemy from without seems to be fairly well under control.

The bane of our daily practice is the enemy from within. We have all felt woefully inadequate in our treatment of the non-infectious conditions of the eye, ear, nose and throat. In a previous editorial[1] we expressed a conviction which had gradually taken form in the course of experience over many years — that in routine practice of our specialties sufficient attention has not been paid to such considerations as faulty nutrition and fatigue. We have been neglecting these important factors. As an example, there is the ever-present problem of susceptibility to colds. Through the years, the usual practice has been to attack local conditions in the sinuses or tonsils, by medical or surgical treatment, or to try out vaccines — either by needle or by mouth. We mentioned that it is neither necessary nor wise to abandon old procedures but that it is necessary to think more and more about these two systemic factors — faulty nutrition and fatigue.

The actual momentary state of the mucosa and other tissues of the body is determined by transitory chemical changes. The patient who is subject to frequent "colds," the untold number of victims of "catarrh" and of sufferers from "hayfever" — all exemplify what we have in mind. We now realize that a hope for the relief of these patients lies in the correction of faulty diet. No doubt we have been guilty also of overlooking the factor of fatigue. Inquiry into the daily lives of these patients often reveals various degrees of exhaustion from overwork, anxiety or disturbed emotional states. Relief often comes from regulation of daily habits or the prescribing of a vacation.

We now know that a lack of the right food produces pathologic change in every tissue in the body. The study and the knowledge of the "intake" — in man and other animals and in plants — have progressed more in the past 25 years than throughout all previous time. It is interesting to conjecture

what may develop in the next 10 or 20 years as we learn to apply these new-found nutritional facts to eye, ear, nose and throat problems. To the busy clinician it may seem a dull and tedious road to travel. It lacks the spectacular; it takes considerable time — and knowledge. However, one does not need to be over-enthusiastic on this subject to subscribe to the following summary of the History of Medicine: Sixteenth Century — Anatomy; Seventeenth Century — Physiology; Eighteenth Century — Pathology; Nineteenth Century — Bacteriology; Twentieth Century (at least to the present time) — Nutrition.

The first half of the "book of our specialties," so to speak, has been well written — concerning the enemies from without; but the second half is now only in the process of composition. In the language of St. Paul: "We are all members of the same body"; the surgeon cannot say to the dermatologist, "I have no need of thee"; nor can the specialist in any field say to the student of nutrition, "I have no need of thee."

It was Shurly[2] who gave us the phrase, "Preventive Otolaryngology." He stated not only that research in the field of nutrition offers an opportunity to raise the vitality of all mankind, but that we may well believe that a genuine practical application of dietetics is indicated in otolaryngology. Shurly gave us this message in 1928!

Stripping away all complexities, we know that the two main foods that are bad and yet in common use throughout civilized countries are white flour and sugar. These are two of the bad products of civilization. The miller takes all the best out of the original flour, gives it to the cattle and hogs—and then feeds us on the refuse—known as "refined" flour. Sugar is of course an incomplete food — providing calories only; it contains none of the other essentials of good food. Just like alcohol, it "dilutes the mixture," as Moose[3] has said. As it were, it reduces the "high octane"; the more sugar or alcohol we take, the poorer the gasoline we have to travel on. While it is true that one can take sugar or alcohol and "get away with it," yet both are bad foods. If the grocers sold no

sugar and no white flour, no one could estimate how great would be the benefit to the health of civilized people. When sugar was scarce during the war, its absence was surely a benefit to those who could not get it. In the second edition of his book on "Nutrition and Physical Degeneration," Price[4] has given the best explanation extant on the dictum: "As a man eats, so is he." The degeneration that can be most readily observed and statistically recorded is in the teeth. For many years Price traveled the wide world, made observations and recorded his findings, particularly of the teeth and dental arches of primitive peoples. As we recorded in a previous editorial,[5] in the Foreword of this original and remarkable book, Hooton appreciates and brings out the new concepts of Price. Hooton recognizes that there is nothing new in the observation that savages, or peoples living under primitive conditions, have excellent teeth; nor is it news that most civilized peoples possess wretched teeth which begin to decay before they have erupted completely. Indeed this has been a matter of grave concern for more than a generation. It seems to Hooton that we have been extraordinarily stupid in concentrating all our attention upon finding out why our teeth are poor, without bothering to learn why savages' teeth are good. He admires Price for being the only person who possessed the scientific horse sense to supplement his knowledge of the probable causes of dental disease with a study of the dietary details which are associated with dental health. Price has found out why primitive men have good teeth and why their teeth decay when they become civilized; but he has not stopped there — he has gone on to apply his knowledge acquired from savages to the problems of their less intelligent civilized brothers. We must admit that if savages know enough to eat the things which keep their teeth healthy they are more intelligent in dietary matters than we. One example of the wisdom of primitive peoples is noted by Price. Special feeding is prescribed for the prospective bride — in some tribes for as long as six to eight months. Price makes clear that the teeth, which are the special objects of his study, are fair criteria for all tissues of the body. Even the process of thinking is as biologic as is digestion; and embryonic brain

defects are as biologic as are club feet. Profuse illustrations contrast the excellence of the teeth and dental arches of primitive peoples with the degenerations seen in those who eat our average modern foods. These observations cover the isolated and modernized Swiss, Gaelics, Eskimos, North American Indians, Melanesians, Polynesians, African tribes, Australian aborigines, Torres Strait Islanders, New Zealand Maoris and Peruvian Indians.

In our daily practice we all would be shocked by the teeth we see, or by the absence of them even in adolescents, were it not that we are so used to seeing this. Some young people, in apparent health, are actually edentulous — giving a somber significance to the old jest, "Mother's teeth will soon fit Katie." No one doubts now that poor food is the main cause of this degeneration. It makes one pause and consider that if white flour and sugar play such important rôles in bringing about these conditions in teeth, what is happening to all tissues in the same individual.

In a stirring editorial Remmen[6] gives another approach to this subject. Politicians tell us that they long to elevate the masses; they proclaim our unhappy state of physical decay which, we are told, is due to a lack of medical care of the people. It is entirely proper that government should be deeply concerned with the health of the people, since they constitute any nation's greatest asset. More fundamental to national vigor, however, than any services which bureaucratic medicine could render is the manner in which the population is clothed, housed, and above all, fed. When our government has accomplished the stupendous task of bringing the national nutrition and housing to tolerable levels, it will surely be found that the incidence of disease will have fallen so markedly as to greatly reduce the need for medical care. Although knowledge of what constitutes adequate nutrition has increased rapidly in recent years, scant attention has been paid to it by our government. Much has been learned experimentally, and the concentration camps and the starving populations of Europe and Asia have furnished ample clinical demonstrations of the effect of starvation in

all grades. The relationship between nutrition and health is becoming more and more clear to the medical profession. Comparatively few Americans are actually hungry. Quantitatively, there is usually enough food — often too much; one may be obese and still undernourished. As to the quality of food, however, deficiency is almost universal. We eat wheat from which the germ and hull have been carefully removed. We feast upon refined sugars, and guzzle oceans of sweetened drinks. We eat vegetables grown on wornout soil which has received a temporary boost by commercial fertilizers; and we let the rich sewage from vast cities run into the ocean instead of returning it to the soil after proper treatment. Erosion, deforestation, "one-cropping," careless irrigation and other sorry agricultural practices have ruined or depleted much of our best farm land. Although such land may produce crops after a fashion, anything grown upon it is lacking to a greater or less degree in essential food elements; this must seriously affect the nourishment and well-being of both domestic animals and mankind. It is a well-established fact, for example, that cows which graze upon land deficient in calcium and phosphorus become less fertile. In brief, the quantity of food available is far less important than its quality. Two eggs may look alike, but their nutritive value depends on what the hens ate. The biochemist may find important differences, and even he may overlook essential but elusive substances. To provide adequate food for all our people is possible, but the task is stupendous and one which only Government can hope to carry to a successful conclusion. A vast program of popular education in the fundamentals of nutrition is most necessary. Farmers must be taught the principles of soil conservation and soil building. Government aid in draining, irrigating, fertilizing and treating soils will usually be needed. The land is a common resource which no individual should be permitted to destroy, either deliberately or through ignorance. There has never been enough meat, fish, eggs, milk, cheese, fruit and wheat available to millions of Americans who have low incomes. As a result, they suffer in health and fall prey to disease. Only a vast governmental program of agricultural modernization and expansion with adequate distribution of

food products will correct the national malnutrition — which causes, or contributes to, more disease and deformity than all the world's doctors can cure. If we as a nation are really sincere in our desire to improve the health of the people, let us first provide enough food of good quality for everyone. And when we have done that, vast slum areas remain to be cleared and replaced with modern healthful homes. Thousands of playgrounds should be built, equipped and supervised. There is also a great need for clothing in many quarters. Then, after our statesmen have amply provided the American people with the three great basic necessities of life — food, clothing and shelter — no one could reasonably object if they took up the subject of regimenting medical care. Until then, first things first!

The most elaborate clinical study of the effect of faulty nutrition and fatigue on the tissues of the sinuses, has recently been made available by Roberts.[7] He laid out a deliberate and detailed plan for the study of patients with non-suppurative sinuitis and, after eight years, gives a statistical report of 4,000 such cases. The results of his treatments have been so gratifying that we may hope that before many years similar methods will be generally used in routine otolaryngologic practice. Roberts outlined a definite pattern of investigation to establish the diagnosis. Sinus trouble of many patients cannot be explained on the basis of infection, deformity or new growth. Nasal surgery, even the absolutely necessary procedures, has fallen into disrepute with the layman and the conservative physician. Roberts found that fatigue was present to some degree in a large majority — actually 87 per cent —of the adult patients with chronic sinuitis, and that some degree of exhaustion was found in 50 per cent. One cannot ignore this voluntary statement of fact by the patients. Statistics showed that 31 per cent of Roberts' large series of cases took a cathartic at more or less regular intervals. Such patients cannot be expected to have healthy bowels. When the lubricating type is used, such as liquid petrolatum, it prevents absorption of "A" and probably of other vitamins in foods. Physicians should never encourage this pernicious

habit. One should think as sharply before prescribing a cathartic to be taken regularly, as one does before giving a prescription for a narcotic. Sugar is certainly not to be recommended as a food. Roberts appraises the autonomic nervous system. If the sympathetic division is dominant, the patient is nervous, irritable, high-strung — people get in his way. He has unlimited energy, and becomes the president of the firm in his early thirties — and then cracks up nervously or has a cardiac breakdown in the late forties or early fifties. His speech is rapid and staccato; beads of perspiration stand out on his forehead, winter and summer; he has a tendency to obesity and baldness; pulse and blood pressure are usually elevated, pupils are somewhat smaller than normal. The patient in whom the parasympathetic division is dominant is the direct opposite, and is much more apt to have allergic symptoms. The sympathetic type needs small doses of iodine and mild acidulation (if in middle life); the parasympathetic type needs thyroid and much more acidulation. Ivy[8] states that gastric acidity slows up after age twenty; achlorhydria increases considerably after forty; and by sixty-five 35 per cent of people do not secrete acid at all. Roberts gives the B Complex freely. To allergic patients he gives "C." During the first month he gives "A" and "D" three times daily. At the end of one month the patient's case is reappraised. In some instances, if the patient follows the food selections, vitamins may be discontinued at the end of six months. The case must be rechecked once a month for several months, or until the food selections and new personal habits become routine. Roberts has found few patients uncooperative; patients are usually so enthusiastic about results that they follow instructions absolutely. When the patient is properly managed, it is not unusual to have the major portion of the symptoms relieved at the end of two months.

Vitamin chemistry affords an intimate vision of a community of inheritance in all living things. Perhaps no one has better expressed what animal and vegetable life have in common than Williams.[9,10] Each of us has about 5 grams of glucose in his bloodstream. Turn where you will, you will find

it in other bloodstreams — and in the sap of plants. Vitamins are the master keys which unlock the stores of vital energy from glucose and other foods, within the cells of all living things alike. Animals came much later than plants and can claim no share in the inventions already present in plants. We who have long boasted as the lords of Creation find that we are really mendicants in Nature's breadline and heirs of the grass of the fields. Wallace[11] outlines the main processes in plant development: Absorption—(intake of water and mineral elements by the roots); Intake of carbon dioxide from the air by the leaves—(the reaction of carbon dioxide with water in the presence of chlorophyll forms sugar and free oxygen); Formation of protoplasm—(the living matter of the plant, consisting mainly of proteins); Respiration—(combination of oxygen with food substances synthesized by the plant); Transpiration—(loss of water by the plant, mainly from the leaves); Translocation—(movement of materials within the plant); and Storage—(storage of reserve products in various organs and tissues). During growth there are continuous processes—the building up of complex compounds of carbon and nitrogen, and the breaking down of these into simple substances. Metabolism comprises these processes, in animals as in plants. Substances are formed such as sugars, starches, cellulose, amino acids, proteins and amides; many plants also produce special products, for example nicotine or caffeine. Mineral nutrients normally enter the plant through the roots. Certain growth-promoting substances, called "hormones" in the plant, include "B_1" which occurs naturally in soil. Whereas in animals the pH range is limited to approximately 6.8 to 7.8, in plants it may extend from 4.0 to 9.0. As in the animal body, lack of needed elements in plants produces effects which make it possible to diagnose nutritional deficiencies.

As Bicknell and Prescott[12] remind us in their comprehensive book, at the beginning of the present century one thought chiefly of carbohydrates, fats and proteins. These, with minerals such as iron and calcium, were believed to supply all the needs of the body. Indeed, at that time the main problem

in human nutrition appeared to be how to provide more animal fat and protein for the poor. This simple idea was accepted until just before the first World War. The discovery of vitamins revolutionized the whole concept, not only in dietetics but also in the study of disease. It soon was realized that many diseases are not caused by infections or toxins, but simply by vitamin deficiencies in the food. Vitamins were discovered at a singularly opportune moment. At the beginning of this century the population was migrating from the country to the towns. Fresh country produce such as vegetables, milk and eggs became too costly for the poor in the cities. Another important factor was the increasing consumption of white flour. Until a few years ago the vitamins were of clinical interest only in the treatment of certain deficiency diseases. It is now realized that, in large doses, they can be used therapeutically in other conditions. Vitamins, like the hormones, can act as drugs. Nicotinic acid has a powerful vasodilator action even in a normal person; this suggests its use in peripheral vascular disease. "B_1" and "C" stimulate metabolism. Wise feeding is the greatest weapon preventive medicine can wield. The children in the big cities of England have been living on diets so deficient in vitamins that three-quarters of them have at some time in their lives had rickets, and one-half have had defective twilight vision. It is probable that 50 per cent of the population of Great Britain, and also of the United States, are receiving insufficient vitamins; particularly is this true of the "B" Complex, since they live largely on white bread, which, far from being the staff of life, is only a broken reed. Civilized man does not instinctively choose a sound diet. In the draft of the youth of the United States, 40 per cent were rejected as unfit for military service; and it has been estimated that one-third of these suffered from disabilities connected with nutrition. Most people in both countries are apathetic; they are content to spend their lives in that shadowed region between good health and frank illness. A lack of "A" may result in corneal vascularization in the rat, indistinguishable from that which occurs from a deficiency of "B_2." Perhaps this vascularization may be caused by an actual deficiency of "B_2." Normally "A" is

present in the tissues secreting tears and in the Meibomian glands; its absence or marked reduction results in keratinization and blocking of the glandular acini. Large amounts of "B_2" are contained in both secretions. A lack of "A" in the tissues, therefore, results in a lack of "B_2" in the tears and Meibomian secretion, and ultimately in corneal vascularization. "A" is found in very few foods; of these the commonest are butter, eggs, milk and liver. The old belief that a dark yolk meant a good egg is undoubtedly correct. The color of the yolk is due to carotinoids, and this is a good indication of the content of "A" itself. Pasteurized milk and dried milk have the same "A" and carotene content as fresh milk, but sweetened and condensed skimmed milk have none. "B_1" is an essential in the transmission of peripheral nerve impulses. The body is unable to store "B_1" for any length of time; the pig, however, seems exceptional in this respect—hence the unusually high content of "B_1" in pork. The concentration of "B_2" in tumor tissue is low in comparison with that of normal tissues; this is in keeping with the view that cancer tissue has a deficient oxidation system. It is known that the energy of the central nervous system is derived entirely from the combustion of carbohydrate; the liberation of energy occurs, not in one large burst, but stepwise by means of several enzyme systems, one of which contains nicotinic acid. A break in the chain of carbohydrate oxidation in the central nervous system may explain some of the mental changes seen in pellagra and nicotinic acid deficiency. "C" appears to be present in all living tissues but fresh fruits and plants are the best sources. It has been supposed that the citrus fruits are the most potent sources of "C," but since chemical methods have become available for its estimation, it has been shown that this is not true. Black currants are three to four times as potent as orange juice; the major portion of the "C" is in the peel—not in the juice. The potato is one of the most important sources of "C." The richest source of "C" is rose hips; the annual yield of rose hips from Alberta, Canada, is sufficient to supply everyone in the United States with 100 mg. of "C" daily. Raw vegetables, such as lettuce and watercress, are important, because they are consumed raw and all the

"C" is available. Until recently it was taught that most of the "C" is destroyed by cooking. This has now been disproved. It is true that the vitamin itself is rapidly inactivated when heated in solution with alkalies, but in natural foodstuffs there are stabilizing factors preventing its decomposition. Fruits and vegetables should not be kept too long before cooking, because "C" becomes oxidized. Less "C" is lost if the boiling is done rapidly with the lid on the vessel. The greatest loss occurs, not through the destruction of "C," but by its extraction in the cooking water which usually is thrown out. The "C" content of fried foods compares favorably with that of boiled or baked foods; frying rapidly seals the food, drives off much of the water, and is quick. Considerable destruction of "C" in fruits and vegetables occurs if the material is shredded or chopped; a shredded lettuce loses 80 per cent of its "C" in one minute. If food cannot be eaten just after it is cooked, it is better to reheat when necessary than to keep it hot; this conserves more "C." The rôle of "C" in the formation of intercellular material in the human subject has been established beyond doubt, but the manner in which it acts is unknown. It has been observed, however, that reticulum and collagen are not formed in the animal with scurvy but when "C" is given bundles of collagenous material form within twenty-four hours. The only good sources of "D" are dairy products and fish. Green vegetables, in spite of all belief to the contrary, do not contain "D." The value of all dairy products depends on what the hens and cows eat and on their exposure to sunlight. Eggs contain nearly three times as much "D" in summer as in winter. The eggs of commerce produced by birds in coops may be almost entirely lacking in both "D" and "A." Butter shows a wide seasonal variation in "D" content. "D" is powerful. All preparations of synthetic "D" are toxic to man in large doses and there is no reason to suppose that "D" of fish liver oils is not toxic, though few human cases of poisoning have been reported—two children died from drinking large amounts of cod liver oil and from prolonged sun baths. Although the rôle of "E" in man is not yet understood, some results have been reported in treating threatened abortion and muscular dystrophy. The

main source of "E" is wheat germ—which is all too seldom used. Wheat germ has been found so valuable for the feeding of livestock that it appears unlikely that it will be left in bread now that the war is over, since attention to the nutrition of pigs is far more lucrative than to that of people. With the removal of the germ, the cheapest and only universally available source of many vitamins is lost. The sorry history of government control of the millers holds out little hope for the future.

Varying degrees of vitamin deficiency call for varied handling. Although this statement is trite, its vital importance is shown by Joliffe[13] in his excellent appraisal of the commoner avitaminoses. There are two main classes of indications for giving vitamins — supplementary and therapeutic. Supplementary quantities of vitamins are indicated when diet is faulty from wilfullness on the part of the patient, or from imposed restrictions, as in obesity, diabetes or infections. Therapeutic amounts, larger dosages, are indicated in true deficiency disease. Joliffe lays great stress on the study of general nutritional build-up as being no less essential than meeting an actual or assumed avitaminosis. He deplores the promotional claims of manufacturers as misleading the less informed public; also the common implication that synthetic vitamins are less effective than those from natural sources. He considers that diets are unsatisfactory in all sections of the country.

As to the butter-margarine controversy, Deuel[14] states that the vegetable oils and fats are practically identical with the animal ones. Fats are the best vehicles for dissolving "A," "D" and "K," and on that score alone fats are essential in food. Fat is also the most concentrated form of energy, having about twice the caloric value of carbohydrate. Today no one questions the digestibility of margarine; in fact, if sufficient "A" is added it has the same value as butter. It has been shown that the fat in human milk resembles margarine more than it does butter. The value of milk is not confined to its fat; it contains proteins of high quality and is an excel-

lent source of certain inorganic salts needed in the diet, especially those of calcium and phosphorus. Milk is a good source of "B_2." One can scarcely view with concern the increasing use of margarine if this means that larger amounts of whole milk become available.

In the opinion of Holmes, Kuzmeski, Jones and Canavan[15] ice cream does not receive due recognition as a valuable source of proteins, fats, carbohydrates, minerals and vitamins. In 1943 the average annual consumption of ice cream was over three gallons per person in the United States. The retail value of ice cream annually consumed in the United States is about one-fourth that of milk, and over twice that of eggs, apples or potatoes. Samples of coffee, maple and vanilla ice cream were assayed for carotene, "B_2" and "C." Comparison with numerous widely used foods shows ice cream to be an excellent source of carotene and "B_2." No "C" was found, probably because of the large amount of air incorporated in commercial ice cream to increase its bulk.

As to food for the aged, Tuohy[16] notes that appetite lessens as age advances; therefore, foods of little caloric value have a place—condiments, broth and relishes. Teeth, gastric acidity and absorptive powers all begin to fail with age. Vitamins, calcium and hydrochloric acid—with balanced diet, can compensate for these losses. When hydrochloric acid is lacking, as occurs with increasing frequency after the forty-fifth year, it becomes a useful medical adjunct in those who have simple diarrhea, or small stools immediately after eating. Multiple avitaminoses are frequently concurrent.[17] The X-ray shows rapid progress of barium through the small intestines; this implies impaired absorption. Hydrochloric acid, by its stimulant action on the pylorus, tends to slow the emptying of the stomach and make for better absorption.

Investigations were made in Russia by Mashkilleison, Benyamovich, Krichevskaya and Shatamova[18] on the rôle of vitamins in skin diseases. They emphasize that the most frequent manifestations of deficiencies are observed in the skin. The lack of "C" produces the characteristic petechiae. Lack of

B Complex causes lesions at muco-cutaneous junctions and in the oral mucosa—cheilosis, stomatitis and glossitis.

Out of some 500 released prisoners of war and internees from Thailand, not less than 100 suffered from marked visual defects. As a result of their captors' barbarity thousands of their fellows had died, under conditions of enforced physical labor beyond their powers, and with a food supply utterly inadequate to sustain life. Ridley[19] found the outstanding ocular manifestations among the survivors to be a loss of visual acuity of varying degrees. Many reported sudden onset, with the maximal disability reached within a single day; in others a state of advanced amblyopia was reached gradually, taking months to develop. Vision was so impaired in some that they could recognize the faces of their friends only when close at hand; they could not see the signals of their guards and thereby incurred punishment. Some experienced photophobia, with vision defective in bright light. Avitaminoses, of various degrees and types, in association with prevalent malnutrition, were linked with pellagra, dry beri-beri and sore tongue; quite a high proportion of the amblyopic also showed nerve deafness. The presence of scotomas was not always associated with visible fundus change; but in many severe cases there was a gray pallor of the temporal half of the disc. Practically all the released prisoners showed kerato-conjunctival abnormality, the limbal capillary plexus being increased, with invasion of the cornea. On admission to hospital the patients received an ample diet and supplementary vitamins; after two weeks the vision of about one-half improved appreciably, especially when they tried to read. In several the density of the scotomas was reduced.

To the United States Naval Hospital in Philadelphia are admitted the blinded personnel of the Navy and the Marine Corps. Eight men captured at Corregidor, who had gone through the long years of captivity in Cabanatuan or Japan, had vision ranging from 1/200 to 10/200 in the better eye. Beam[20] found the outstanding pathologic change to be atrophy of the optic nerve, with a central scotoma in each eye.

He attributes the degenerative change to the conditions of dietary deficiencies, chemical and bacterial intoxication, and strenuous enforced labor. The outstanding deficiency was that of "B_1," the need of which was great and the supply negligible. Nerve deafness was found in seven of the patients, in association with the optic atrophy.

Recalling Wilson's[21] summary of medical problems among American prisoners of war at Cabanatuan and Santo Tomas, there comes a complementary report of conditions in the former camp as seen by the dental service. Fields[22] reviews the organization of the dental department and the work done which was largely along the lines of conservation. More than 50,000 sittings were recorded. The progressive physical impoverishment created changes in the oral tissues, so that many dentures became unserviceable. Various manifestations of avitaminoses, traceable to pellagra, beri-beri and scurvy, were observed in the mouth, but the food had been so inadequate that Fields found it impossible to differentiate between one vitamin deficiency and another.

Offsetting the current trend towards emphasis on nutrition comes a review of dental disease showing the importance of heredity. In the Colorado River Relocation Center, 5,400 persons of Japanese ancestry were examined by dentists, working under the supervision of the United States Public Health Service. The findings are tabulated by Klein[23] for parents and children, the calculation being based on the number of teeth decayed, missing or filled (DMF being the accepted symbol); according to whether the fathers' and the mothers' DMF ratings were low, medium or high, the offspring was found to show a fairly constant ratio; for example, for the ages 10-14 years daughters of the mothers and fathers with low DMF rates average 3.4 DMF teeth, and the daughters of parents with high DMF rates at the same ages average about 6.6 DMF teeth. There is a marked and consistent tendency for children to reflect, in their own caries rates, the DMF tendency of the parents.

In an orphanage in the Province of Quebec, 171 children,

aged between 8 and 13 years, were chosen for a six months' dental survey by Strean and Beaudet.[24] Some received tablets containing calcium fluoride (3 mg.), "C" (30 mg.) and "D" (400 units); a second group got tablets of calcium fluoride (3 mg.) alone; a control group received no tablets. Additional studies were conducted outside the orphanage. While no group showed an absence of dental cavities, the results suggested that fluorine in combination with "C" and "D" is of distinct value in keeping down the number of cavities appearing in the permanent teeth of growing children.

The value of fluorine as an inhibitor of dental caries has been under appraisal for about eight years. Jay,[25] after a study of nationwide statistics, deduces that the ingestion of fluorine is of the greatest benefit when it is taken in the first eight years of life. Where fluorine is a natural content of domestic water the caries rate tends to be considerably lower than where the water is fluorine-free. The hardness or softness of the drinking water is not found to bear upon the prevalence of dental caries. Two long-range studies are now in progress—at Newburg, N. Y., and Grand Rapids, Mich.—with fluorine being added to the water to give approximately one part per million; the trends will very likely not be discernible before 1950 and possibly later. Fluorine tends to inhibit the activity of oral Lactobacilli and very probably other bacteria in the mouth, but how it operates is a matter of speculation. In fluoride areas the Lactobacillus count in saliva is very seldom found at a high level. A possible substitute in rural areas for the use of fluorine in drinking water is internal medication by doses of sodium fluoride, about 2 mg. daily.

"A"

Experiments by Warkany and Schraffenberger[26] indicate that "A" is essential to the development of normal eyes. Female rats received a preparatory diet which contained enough carotene for growth and maturation, but prohibited a storage of "A." When mature, the rats received a diet completely free of carotene and "A," and then were bred to normal rats of the same strain. Within one or two weeks the

mothers showed signs of "A" deficiency and their young presented, among other malformations, many ocular defects. Histologic sections of all the eyes showed a fibrous retrolenticular membrane in place of the vitreous; there were colobomas, eversion and abnormal structure of the retina, poor development of the iris and of the ocular chambers, defects of the cornea and of the conjunctival sac, and a lack of fusion of the lids. When the maternal diet was supplemented with "A" during pregnancy the eyes of the young were normal.

Changes in the tissues of the eye of rabbits deprived of "A" do not appear early in the progress of the deficiency. Mann, Pirie, Tansley and Wood[27] found that the Plasma-"A" values sank to about 10 per cent or less of the normal before any sign of deficiency could be observed with the slit-lamp. The earliest detectable change was superficial, a few central cells of the corneal epithelium becoming opaque and greasy in the course of a metaplasia into squamous keratinized cells. No corneal ulceration developed. With extension of this corneal change came an apparent smoky discoloration of the bulbar conjunctiva. Subsequently appeared an actual keratinization of the conjunctiva. Happily, the investigations led to the conclusion that even the severe changes due to "A"-deficiency, on treatment with "A" prove in shorter or longer periods to be reversible—recovery being complete.

From lack of "A" arise pathologic changes in many tissues. What affects the retina may also affect the skin. Poor diet in children from the first year of life on, and in adults under wartime conditions or other near-famine states, causes in the skin cornification in varying degrees, with many dermatological names; and in the eye, subnormal readings of the biophotometer. When "A" was given in daily doses of 100,000 to 300,000 units to the children whose cases are reported by Lehman and Rappaport[28] there was a prompt retinal response and a skin improvement, though longer delayed.

Applying for enlistment in the Navy, a young volunteer was found to be physically in excellent condition, with the

single abnormality of extreme miosis. Gerstle[29] found that during 40 days the boy had taken approximately 7,000,000 units of "A." On his discontinuing the taking of the vitamin, his pupils became normal within five days.

In children with no digestive trouble, Cienfuegos[30] reports normal absorption of "A" when given by mouth, whereas when injected into muscles little or none was absorbed.

B COMPLEX.

The classical signs of "B" deficiency — cheilosis, nasolabial seborrhea, nervousness, polyneuritis and associated physical changes—are attributed to nutritional errors. For their correction the B Complex or its several components are given, often without clear thought of how and where vitamins work. In cases of diabetes with "B" avitaminosis, Biskind and Schreier[31] lay the blame on a liver inactivity. In a consideration of the function of the liver in diabetes, they link a shortcoming in its maintenance of carbohydrate balance with the evidences of B Complex deficiency. It is striking to read their quotation from Osler's Modern Medicine published in 1908: "The tongue in diabetes is large, red, 'beefy,' and bordered with a fissured margin . . . ," the typical description of glossitis in pellagra. The use of B Complex in high therapeutic dosages does away with the avitaminosis, enables the liver to respond to the patient's own insulin, and actually cuts down—in some cases abolishes—the need of taking insulin. In a group of 94 diabetics only two proved refractory to this nutritional mode of treatment; both had very severe indications of prolonged "B" deficiencies and had no doubt suffered irreversible tissue changes or else were in need of nutritional factors as yet unknown. Of the 92 patients who were benefited, 14 who had taken an average daily dose of 41 units of insulin were now able to keep themselves free of glycosuria on an average dose of 18 units; 16 patients were able to discontinue their insulin. A general improvement of health was the rule, being shown not only in the carbohydrate metabolism but also in the manifold lesions traceable to the deficiency of "B" factors. The nutritional factors given

orally were usually administered in the following daily amounts, in divided doses after meals: thiamine 36 to 45 mg., riboflavin 21 to 36 mg., niacin amide 200 mg. (occasionally increased to 500 mg.), pyridoxine 3 mg., calcium pantothenate 12 to 27 mg., choline 210 mg., inositol 27 to 150 mg., L. casei (folic acid) factor 60 to 280 micrograms; these vitamins were derived in part from crystalline material and in part from brewers' yeast extract, 80 per cent alcohol-insoluble liver extract, desiccated whole liver or combinations of these (the inositol and folic acid were derived solely from the natural sources); 225 mg. accorbic acid was often included.

Whether given by mouth in tablet form or by the syringe in solution, "B_1" is still "B_1." Reingold and Webb[32] give an arresting warning against the widespread idea that intravenous or intramuscular use is without risk. The first note of alarm was sounded by Mills[33] in 1941 when a fatal case was reported; the authors now present a second fatality. A woman aged 25 received in three weeks three intravenous injections, each time 1 cc. of a solution containing 100 mg. of "B_1." Seventeen days after the third injection the patient, who complained of being jittery, received her fourth dose. Almost immediately she became dyspneic and cyanotic, and in spite of an injection of epinephrine lost consciousness and died. This resulted from a series of conditions—*1.* preparatory contact, *2.* appropriate period of latency, *3.* the subsequent or eliciting contact. Other instances quoted are of anaphylactic reactions, in which the patients showed alarming distress, fortunately without tragic outcome. "B_1," like other foodstuffs, is best taken by mouth. If there is definite reason to administer it by injection, an intradermal test should first be made.

In the administration of "B_1" through a period of many months Laws[34] believes that the intervals between treatments should not be lengthened to such a degree as to risk anaphylaxis. He reports the case of a woman aged 72 who received 25 mg. subcutaneously daily for 10 days, then weekly for about six months. In the fourth month she noticed that sneez-

ing attacks followed soon after receiving "B_1." After an extended interval between doses—10 days—the next injection brought on a severe reaction with urticarial wheals, cyanosis and dyspnea, the reaction lasting five or six hours. Laws reasons that, in this case, a seven-day period did not exceed the latent phase in the development of anaphylaxis, but that the increase to a 10-day interval changed the picture and precipitated the anaphylactic reaction.

In some people—the percentage of the whole population must be extremely small—a sensitivity to "B_1" is built up during long continued courses of its administration. Schiff[35] encountered such a case in a woman of 49 suffering from a mild form of Reynaud's disease and sciatica. She received in nine months 58 intramuscular injections of 25 mg. of "B_1." There were intermissions of 8 to 30 days on account of unfavorable reactions, nausea or vomiting. She reported, also, after the final and most alarming reaction, that she had had bouts of sneezing following the preceding three or four injections. Her collapse on the occasion of the last injection was immediate; she vomited, voided involuntarily, broke out in a cold sweat, became pulseless and stopped breathing. She received epinephrine intravenously and subcutaneously, and caftein with sodium benzoate. These stimulants and continued artificial respiration resulted about half an hour later in restoration of breathing and normal heart action. Scratch tests in the month following gave positive reactions for "B_1" in commercial preparations with preservatives and a pure aqueous solution, but negative reactions for the preservatives. The shock, in Schiff's opinion, was strongly suggestive of sensitivity to "B_1."

Decrying the common practice of giving for long periods amounts of "B_1" in excess of bodily needs, Mills[36] describes the effects in two women of daily doses of 10 mg. and 17 mg. respectively. Both presented symptoms like those of hyperthyroidism — rapid pulse, tremor and nervous irritability; and, in both cases, on stopping the use of "B_1" the patients' toxic manifestations came to an end. The older woman

resumed the use of "B_1" after one week, taking 5 mg. a day, but after four and a half weeks had to stop taking it. The same problem was propounded by Mills[33] in a discussion of Joliffe's paper on the use of vitamins in neuropsychiatry. Instead of two patients, Mills reported on many whom he had seen in Panama and Cincinnati. These cases included one fatality in which at autopsy were found sub-pial ecchymoses, encephalo-malacia and perivascular hemorrhage; and several near-fatal collapses. These severe reactions all resulted from intramuscular injections of "B_1" in 10-50 mg. doses. To Mills they seemed definitely allergic; he draws no distinction between toxicity and allergy.

"B_2"

A finding of practical importance is reported by Stamberg and Theophilus.[37] "B_2" readily undergoes photolysis. Milk boiled in a light room loses much more "B_2" than milk boiled in the dark. Bottles of milk exposed to sunlight lose from 30 per cent to 54 per cent of "B_2"; the loss is greater upon longer exposure to light. Since milk is probably the commonest source of "B_2" for children as well as for most adults, milk should be kept from exposure to sunlight. The photolysis in raw milk is greater than in pasteurized milk, and least in homogenized milk.

To 300 patients with ocular disease whose diets had been deficient in "B_2," Spies, Perry, Cogswell and Frommeyer[38] gave "B_2" intravenously. Within 48 hours all patients reported some subjective improvement; in most the caliber of the dilated vessels in the eye diminished; and there was a striking decrease in the photophobia and corneal ulceration. Although many had irreparable eye damage, they were relieved of pain and their vision improved. Seventy-two per cent were able to return to work after months and years of idleness. After this, most of the 300 patients went back to their inadequate diets with the result that 251 had recurrences, some soon, some after a passage of time. In 49 there was no clinical evidence of recurrence, although their diets had not been improved.

In rats fed upon "B_2"-deficient diet the first ocular changes are conjunctival edema and congestion. About a week later the vessels at the limbus become much congested and the cornea is marked by faint nebulae; the edema and opaqueness of the cornea continue to increase in intensity, then subside; the final state is that of extensive vascularization, often across the center. Very similar conditions were observed when the diet was "A"-deficient, but the end result was xerophthalmia, loss of corneal luster and desquamation of masses of conjunctival epithelium from the lids. Bowles, Allen, Sydenstricker, Hock and Hall[39] describe the variation in vascularity in the eyes of 500 rats, and show illustrations of corneal lesions typical of "A" and "B_2" deficiencies.

FOLIC ACID.

For the past 10 years the attention of both laboratory workers and clinicians has been drawn to the value of a substance present in liver extract and in yeast, and of established value in macrocytic anemia. The types of anemia thus benefited include the nutritional, the pernicious and those found in sprue, pregnancy and certain wasting diseases. The substance has received different names, vitamin "M," Lactobacillus casei factor and folic acid. Spies,[40] in whose laboratory at Birmingham prolonged study has been done, begins his discussion of the subject with words of regret that the folic acid benefit is not to be looked for in leukemia, aplastic and other anemias of undetermined origin. He includes in his report the results of a special study of 45 patients and gives in detail the blood pictures of 27 at the beginning and the end of treatment with folic acid; the methods of administration were oral, intramuscular and intravenous, and the amount given ran from 20 mg. to 200 mg. daily. In almost all 27 cases there was a substantial increase of red cells, hemoglobin, and reticulocytes.

By feeding monkeys a diet deficient in the B Complex it is possible to induce malnutrition, anemia and leukopenia. The addition to the diet of liver extract or dried brewers' yeast

prevents or corrects these results. Langston and his co-workers published their investigations in 1936. Doan, Wilson and Wright[41] have followed up this work and by confirming the laboratory studies and applying their findings clinically are able to point to a blood-builder as effective as liver or yeast, without their disadvantages. Folic acid, a synthetic L. Casei factor, not a component of the B Complex, and first termed vitamin "M," is found in both liver and yeast. It may be given intravenously, 2.0 mg. daily for a lengthy period, or in increasing doses up to a maximum of 20 mg. per dose, without evidence of a sensitivity. The work done on monkeys indicates that folic acid fills a specific requirement for normal bone marrow function.

The manifestations of sprue in man are similar to those of an "M" deficiency in the monkey. It has been demonstrated that the administration of "M" is curative for "M" deficiency in monkeys. The availability of synthetic "M" (lacto bacillus casei factor, or folic acid) prompted Darby and Jones[42] to give this material in two cases of non-tropical sprue. Striking clinical and blood changes occurred rapidly during intramuscular injections of 15 mg. of synthetic folic acid. Although remissions do occur in sprue the results obtained suggest that folic acid may have a specific effect.

In line with the work cited above is that of Darby, Jones and Johnson,[43] who report on the activity of Lactobacillus casei factor in three cases of sprue. In these patients the characteristic soreness of the tongue disappeared, in only four days of treatment. The authors propose that the group of substances now known by definite names, L. casei factor, folic acid and "M," be termed the "vitamin M group."

In laboratory tests, purified diets containing succinyl-sulfathiazole led to the development in rats of leukopenia, granulocytopenia and, less frequently, anemia. Endicott, Daft and Ott[44] showed that L. casei factor ("folic acid") corrects or prevents these dyscrasias; the response of the depleted bone marrow is a rapid regeneration and a return to normal—after a temporary overcompensation.

"C"

For twenty-two months a rigidly controlled "C" experiment on a human subject was carried out by Pijoan and Lozner.[45] Beginning with a state of "C" saturation, the man consumed no "C" for seventy days, then 5 mg. daily for fifty days, then 10 mg. daily for eighty days, and finally 18-25 mg. daily for the remaining fifteen months. At no time did the subject experience objective or subjective clinical symptoms.

Noting that Rush[46] had shown a lack of calcium, "B_1," "B_2," niacin and "C" in the diet of soldiers, Golden and Schechter[47] made a study of the "C" status of troops in Dutch New Guinea—a survey of nearly 700 subjects in the Army. The average length of tropical service was not less than five months, which allowed sufficient time to estimate the value of the diet. It is generally accepted that recent dietary intake of "C" is reliably indicated by its plasma level. A level below 0.6 mg. indicates less than the optimal intake of "C"; a level of 0.4 mg. is considered definitely subnormal. When the plasma level drops below 0.3 mg., the level in the white cells begins to diminish; this indicates that tissue stores of "C" are approaching the pre-scurvy zone. From the data obtained it appeared that many troops had inadequate levels of "C." About one-half of the men and one-third of the women did not show a satisfactory margin of safety. This lack is not explained by the heat; the amount of "C" in sweat is negligible.

Following an earlier study on the mineral content of breast milk, Winikoff[48] investigated the "C" content of breast milk of women in the Melbourne area. For the normal process of the child's dentition "C" is essential. Infants who are artificially fed require supplemental "C," as cow's milk has a low "C" content. This study revealed that in Melbourne the milk of nursing mothers, with a "C" content ranging from 3.0 to 4.0 mg. *per centum*, was poor in comparison with "C" contents reported in other countries. Setting 20 to 40 mg. of "C" as a daily requirement, she found that the average "C" content of breast milk provided this amount only during the first

and second months of lactation. She considers it urgent to give "C" as a supplement to breast feeding as early as the first few weeks of life.

Having previously reported[49] 100 cases of vasomotor rhinitis in many of which improvement followed large doses of Calcium—"C," Ruskin[50] made a further study and concluded that "C" plays a valuable rôle in the treatment of nasal allergy—if it is given in large doses. The results are advantageous to allergic patients, with or without desensitization—in fact, this "C" therapy proved superior to desensitization. He regards allergic disturbances as being related to nutritional deficiencies, primarily that of "C."

In twelve cases of corneal ulcer with hypopyon Summers[51] used drops of penicillin locally at intervals of one to three hours. In some cases he gave "C" intravenously, 500 mg. daily. Three cases were regarded as hopeless from the start, but in only one was it necessary to remove the eye. All the other patients had complete recovery except for scars. The absorption of the hypopyon did not keep pace with the healing of the ulcer; however, it was definitely hastened by the use of "C."

"D"

It is made clear by Bauer and Freyberg[52] that "D," especially when given in large doses, should be considered as a potentially toxic drug. Its administration should therefore be carefully supervised. The patient should be kept under close observation. Symptoms of toxicity are not uncommon, such as anorexia, nausea, vomiting, diarrhea (sometimes with bloody stools), polyuria, muscular weakness, lassitude, headache and at times profound depression. It is important to detect these earlier symptoms and to reduce the dose or withdraw the drug to avoid the danger of more serious toxic effects. The most severe effect is scattered calcification, which may lead to death. This is rare in the human being, especially in the adult, but it may occur. This should make one cautious. For many years it has been shown that experimen-

tal animals can be killed by large doses of "D"; the outstanding finding is diffuse calcification, involving chiefly the arterial system and the kidneys.

Persons who take vitamins of all types, indiscriminately and in large amounts, are warned by Bauer[53] that "D" can cause death. Adding to previously reported fatalities, he reports the death of a woman who took "D" without a doctor's prescription, seeking relief from arthritis. She took 100,000 to 500,000 units daily for a year. Autopsy showed calcified deposits in kidneys, heart and arteries. The lay person who gives "D" to himself is usually ignorant of the toxic symptoms—loss of appetite, nausea, vomiting, diarrhea, muscular weakness, soreness, fatigue, frequency of urination and headaches.

The danger of taking "D" in very large amounts has been reported previously in our series.[54] Specific indictment is made by Freeman, Rhoads and Yeager[55] against a particular preparation—Ertron. Two adult patients had taken this substance in very high dosages, one receiving 300,000 units of "D" daily for three months, the other 50,000 units up to 300,000 units daily. Both patients suffered from severe manifold symptoms—anorexia, skin eruptions, conjunctival congestion—and organic involvements of the kidneys and prostate, and also subnormal temperatures. A third case is that of a young boy, a victim of arthritis who was under observation before, during and after the use of Ertron (100,000 units of "D" daily). During the period of its administration he developed albuminuria; his serum calcium was not elevated, an evidence that the deposition of calcium in the kidneys may take place without hypercalcemia. The article is beautifully illustrated; the X-ray pictures show dense calcifications in and around the shoulder joint, and, in another case, marked calcifications in the prostate and above the hip joint. Subsequent pictures show disappearance of these calcifications after the discontinuance for six months of the ingestion of Ertron and of milk.

The menace of large doses of "D" and the likelihood of

regional calcifications is discounted by Wolf[56] who has for five years made use of the vitamin in single massive quantities—300,000 units to 600,000 units. Human beings can tolerate as much as 20,000 units of "D" per kilogram of body weight given daily for indefinite periods without intoxication. In the presence of pyogenic infection of the kidneys or other renal pathology "D" may prove definitely toxic and lead to calcification of the arteries, kidney tubules and other tissues. Wolf advocates the massive dose technique in young children as a means of averting rickets, treating tetany and dental caries, and avoidance of allergic sensitizations. Subject to variation his routine is to give 300,000 units every three months; or 600,000 units every four months; or the same amount once or twice during the winter months. Parenteral injection is required only in exceptional instances.

"K"

A fall occurs in the prothrombin concentration of infant blood during the first few days of life; this drop begins about the second and continues to about the fifth day of life. Bleeding developing during this period has been attributed to the lowered prothrombin level. Some authors maintained that the incidence of retinal hemorrhages could be reduced by giving "K" to the mother during labor; some have held that the "K" could actually eliminate this hemorrhage. To study this problem Falls and Jurod[57] examined the fundi of 432 infants, twenty-four to forty-eight hours after birth. The prothrombin time was checked in infants from mothers who had received no "K" and from mothers who had received "K." In following the retinal hemorrhages from day to day it was noted that their absorption occurred very rapidly, leaving little or no trace; in fact it was necessary to observe the majority of the fundi within the first twenty-four hours in order to be accurate regarding the true incidence of retinal hemorrhages. If a lack of prothrombin were the sole factor, one would anticipate a persistent and possible increase in the number of hemorrhages after forty-eight hours, because the decrease in the prothrombin level of infant-blood occurs after

the first day. It was concluded that a lack of prothrombin is not of much importance in the etiology of retinal hemorrhage. This study offers support to the concept that "K" raises the prothrombin level and may play a rôle in prevention of certain hemorrhages. Issue is, however, taken with the reported findings that antepartum or intrapartum administration of "K" materially reduces the incidence of retinal hemorrhages.

INTRA-UTERINE FACTORS ("K," Rh AND RUBELLA).

In an impressive way Potter[58] has correlated three recent discoveries: *1.* the Rh factor and its relation to erythroblastosis fetalis, *2.* "K" and its relation to hemorrhagic disease of the new-born and *3.* the recognition of the harmful effect on the embryo of the rubella infection during the early months of pregnancy. It has been definitely established that maternal immunization to the Rh factor results in a hemolytic anemia in the fetus. All individuals are born Rh plus or Rh minus, and remain the same throughout life. The Rh character is inherited in a simple Mendelian manner, with the positive gene dominant over the negative. Consequently it is possible for two Rh plus individuals to have an Rh minus child, but impossible for two Rh minus individuals to have an Rh plus child. When it was found that "K" can decrease the prothrombin time, many thought that hemorrhage as a cause of death in the new-born would become a thing of the past. Then came dissenting voices. Reports indicated that there was a relatively small decrease in mortality following the administration of "K"; at the present time most of the evidence indicates that little saving of life in the new-born can be expected from the routine administration of "K" to either mother or infant. Occasional infants showing spontaneous bleeding and prolonged prothrombin time will cease to have hemorrhages when "K" has brought the prothrombin time to a normal level.

As to the congenital defects due to rubella, from South Australia in 1943, Swan, Tostevin, Moore, Mayo and Black[59] reported 49 cases occurring during pregnancy; 31 of these women gave birth to children with various defects. With but

two exceptions, these 31 mothers had contracted rubella within the first three months of pregnancy. Of the 31 children, seven were born deaf-mutes. In a further report in the following year,[60] in 10 out of 12 cases the rubella infection occurred within the first three months, and in all 10 the offspring had abnormalities. In the two other instances the mothers had been infected during the fifth and sixth months; their babies were completely normal. Of the group of 10, five were deaf-mutes. The seven deaf-mute children of the first series were two and one-half years old when the records were made. In the later group of 10, the five who were deaf-mutes were studied when over three years of age. Another child died at the age of six and one-half months. The mother had had rubella during the first month of pregnancy. The infant had congenital cataracts, patent ductus arteriosus, was undersized and was thought to be deaf. Serial sections of the internal ear showed: the VIII Nerve and spiral ganglion appeared to be well formed; the bony spiral lamina and the basilar membrane were readily demonstrated; the stria vascularis contained relatively few blood vessels and was shallower and less cellular than normal; the membrana tectoria was rudimentary; Reissner's membrane could not be found; and the semicircular canals, bony and membranous, were well formed.

Additional evidence of the devastating effect of maternal rubella on the embryo in the earliest months of pregnancy is furnished by Carruthers;[61] to the foregoing he adds 18 cases of deaf-mutism in infants, traceable to rubella in the mother. Additional stigmata were stunted physical growth, microcephaly, strabismus and congenital heart defects. The deafness was not absolute in some of the infants studied, but their speech function was practically nil.

Because of the great interest in this new problem of the effects of rubella during the early months of pregnancy, Fox and Bortin[62] made a statistical study. Rubella has occurred in Milwaukee in epidemic proportions approximately every ten years. In a survey of the period 1942, 1943 and 1944, there were 22,226 cases of rubella reported. Of 152 married

women whose cases were investigated, 11 were pregnant at the time they had rubella. The disease occurred during the first two months in five; during the second to fourth month in four; and in the seventh and ninth months, one each. One still-birth occurred among the 11 cases; one woman had twins, both normal; one woman, after a normal pregnancy, had given birth to a child with congenital cataracts, whereas on a subsequent pregnancy she had rubella in the second month, and bore a normal child.

PROTEIN HYDROLYSATE.

The problem of malnutrition is very largely one of protein deficiency; nitrogen loss and nitrogen intake determine nitrogen balance. In 1939 and 1940 pioneer work was done in the application of amino acids to clinical use—in the form of a protein hydrolysate. Tui[63] reports on the high value of protein hydrolysates in different conditions; he finds that in general the nitrogen values of the various diets available in the hospital kitchen are insufficient in their supply of nitrogen — in fact the amount of nitrogen present in even the higher protein diet would not suffice to make up for nitrogen loss in most cases of disease and injury. Body weight may be a very poor index of the degree to which physical powers sink, on account of abnormal extracellular fluids. This is abundantly shown in the many reports of hypoproteinemia that have reached us from prison camps in both the Occident and the Orient. In civil practice there is similar but less spectacular failure of health. Tui discusses the subject of protein hydrolysates—not from the chemical but from the clinical and nutritional point of view; he gives in sufficient detail reports of cases in which these protein "building-stones" served to reduce periods of convalescence by as much as 75 or 80 per cent.

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The Research Study Club,

2507 Washington Blvd., Los Angeles 16, Calif.

MISSISSIPPI VALLEY MEDICAL SOCIETY MEETS AT BURLINGTON, IOWA, NEXT OCT. 1-2-3.

The Twelfth Annual Meeting of the Mississippi Valley Medical Society will be held in the Municipal Auditorium at Burlington, Iowa, Oct. 1, 2, 3, 1947, under the presidency of W. A. Sternberg, M.D., F.A.C.S., of Mt. Pleasant, Iowa, a trustee of the Iowa State Medical Society.

At the November meeting of the board of directors the following officers were elected: President-elect, W. O. Thompson, M.D., F.A.C.P., Chicago, Ill.; first vice-president, B. J. Dierker, M.D., F.A.C.S., Ft. Madison, Iowa; second vice-president, J. F. Ross, M.D., F.A.C.S., Golden, Ill.; third vice-president, D. L. Sexton, M.D., F.A.C.P., St. Louis, Mo; secretary-treasurer, Harold Swanberg, M.D., F.A.C.P., Quincy, Ill.; accounting officer, Ralph McReynolds, M.D., F.A.C.P., Quincy, Ill. The executive committee which will have charge of the Burlington meeting comprises Dr. W. A. Sternberg, Dr. W. O. Thompson, Dr. D. L. Sexton, Dr. Harold Swanberg and Dr. J. C. McKitterick.

MY MILESTONES.

HARRIS P. MOSHER, M.D.,
Marblehead, Mass.

Continued from the November, 1946, issue of
THE LARYNGOSCOPE.

PART II.

THE SURGERY OF THE ETHMOIDAL LABYRINTH.

I gave many years to the study of the anatomy of the ethmoidal labyrinth, and in simplifying for myself the intranasal ethmoidal operation. More students came to my Post-Graduate course on account of the ethmoidal labyrinth than anything else. Frank variations in the structure of the labyrinth did not interest me especially. They were pretty to draw, but it was what might be called the constant or normal anatomy which seemed to me important. Applied anatomy was what I was after as much as possible.

Three points in connection with the ethmoidal labyrinth seem to me to have the most applied value. They are: Where an agger nasi cell is present, it is the lowest and most accessible part of the labyrinth anteriorly; a fronto-ethmoidal cell is often in series with the posterior wall of the frontal sinus. When it is infected, and it often is in chronic infection of the frontal sinus, failure to open and drain it is one of the causes of the failure of the radical frontal sinus operation. An X-ray taken in the Water's position will show the presence or absence of this cell. Usually the ethmoidal labyrinth is wider behind than in front so that the operator has more room posteriorly; however, it is not uncommon to have a flat, narrow labyrinth the same width throughout.

I cannot imagine a surgeon attempting the exenteration of the ethmoidal labyrinth without adequate training on the

cadaver. Even with this training the operator may sometimes lose his way and become confused. If this happens he should at once stop the operation and complete it at another time. This will embarass him less than perforating the cribriform plate.

For the beginner the external ethmoidal operation is the safer. Lynch's technique of removing almost the whole of the os planum in doing the external operation gives a much better approach to the posterior ethmoidal cells.

THE TEAR SAC OPERATION.

In 1913, I attended the International Otolaryngological Congress in London. West came over from Berlin to report on 60 cases of his intranasal tear sac operation which he had done there. His operation had been known for some years, that is, it had been in the medical literature. For me, his was the outstanding paper of the meeting. On returning home I hastened to get a seat on the band wagon by devising and publishing an intranasal tear sac operation of my own. I used it on some six cases with fair results, but came a cropper with the seventh. I got an orbital abscess at the inner canthus of the eye which resulted in a vicious ulcer of the lower half of the cornea. For weeks I followed Dr. Lancaster on his daily ward visit to the patient as he tried to check the progress of the ulcer. He finally managed this, but not until the lower half of the patient's cornea was opaque. I had a dim knowledge of Toti's external tear sac operation and re-read it. It appealed to me on fundamental surgical principles, that is, it was a straight shot and an operation by sight. I unconsciously modified his technique and made the operation simpler. The operation as simplified has been known for years as the Mosher-Toti operation and has a long record of successful operations to its credit.

Where the operation has given a poor result it often has been due to the fact that the operator did not remove enough of the anterior ethmoidal cells. At the Infirmary, Ophthalmo-

logical House Officers have been allowed to do the operation without previous training in the anatomy of the ethmoidal labyrinth and the method of removing the anterior ethmoidal cells by the external route and by sight. Their results were poor.

The crucial part of the modified Toti operation was a generous removal of the anterior ethmoidal cells and the excision of the whole of the nasal wall of the tear sac. I did not attempt to anchor the remaining part of the sac by stitches, but depended upon a large opening into the nose and a sufficient removal of the anterior ethmoidal cells to prevent granulation tissue from sealing off the opening of the lachrymal duct.

Dr. Paul Chandler, now one of the Senior Eye Surgeons at the Infirmary, after gracefully submitting to cadaver work on the ethmoidal labyrinth at the medical school, followed my technique for some years with satisfactory results. If I quote him correctly, he got 80 per cent cures, both of pus and tearing. Of late, he feels that he has improved upon the technique of the modified Toti operation by making a T shaped incision in the inner wall of the sac (the bar of the T being at the top), and anchoring the sac open by two sutures in both edges. He feels, and his results seem to justify him, that the spread out flaps of the inner wall of the sac minimize the formation of granulation tissue from the edges of the bone opening into the nose. By this modification of the operation, his cures are now well over 90 per cent. Many rather complicated methods of suturing the edges of the outer wall of the sac have been advocated. Judging by the results of the modified Toti operation, it seems to me that they have proven unnecessary. Dr. Chandler's method of suturing rather appeals to me. In all tear sac operations, I still feel that a high deflection of the septum should be corrected, and at the operation on the tear sac, good residual space should be obtained by a generous removal of the anterior ethmoidal cells plus the removal of the anterior end of the middle turbinate.

THROMBOSIS OF THE CAVERNOUS SINUS.

In what now seems to me the very dim and distant past I developed a cadaver operation for reaching the cavernous sinus in cases of septic thrombosis. It was essentially an enlargement of the infraorbital fissure of the orbital face of the sphenoid bone and a curette plunge into the sinus. The operation called for the removal of the eye as the sight is usually lost should the patient recover. The removal of the eye was the great stumbling block in the procedure. Patients or relatives of the patient are slow to consent to this; however, in New Orleans, an otolaryngologist had three successes with the operation on Negro patients. I tried the operation but once on the living. The patient was moribund and died within a few hours. This case was experimental surgery on my part. This seldom results to the credit of surgery as a whole or to the individual credit of the surgeon.

Since the time of this operation I have accumulated the orbital contents from three autopsies on cases of cavernous sinus thrombosis. On histological examination of these the orbit was found to be studded with abscesses especially posteriorly near the inferior orbital fissure. These findings suggested that it might be worth while in full blown cases of cavernous sinus thrombosis to remove the posterior half of the os planum by the orbital route and explore the apex of orbit, hoping to find a lake of pus into which the cavernous sinus was draining. I have never tried the procedure and it remains a theoretical operation and probably will continue to do so, especially in these days of chemotherapy.

ORBITAL ABSCESS.

In my early days at the Infirmary orbital abscess was the exclusive property of the ophthalmologist. His method of treatment was repeated puncture by his deft Italian hand above and below the globe of the eye. In weeks or months the abscess healed. We now know that it was healed mostly by nature, not by the efforts of the surgeon. When rhinologists had a chance to see these cases, they suspected that

the fundamental pathology was in the ethmoidal labyrinth, and external operations soon proved they were right. The abscess was due to infection in the ethmoidal labyrinth which had extended to the orbit and was using the orbit as a drainage outlet. It is not uncommon to find a frank break through into the orbit at the posterior part of the ethmoidal labyrinth. In orbital abscess in the adult if there is fixation of the globe of the eye, the external ethmoidal operation gives a rapid cure by removing the basal pathological condition.

In children the condition is not quite the same. The infection again originates in the ethmoid and spreads to the orbit, producing edema, but as a rule, no pus. In most cases it subsides of its own accord or with proper nasal medication. Only rarely is the external ethmoidal operation necessary.

THE PHARYNGO-MAXILLARY FOSSA.

The pharyngo-maxillary fossa is the great hiding place for pus, especially pus high in the neck. It has been abundantly described in the literature, overdescribed it seems to me from the standpoint of applied anatomy. With a blow pipe and strong lungs one can make innumerable compartments in connection with the fascial planes of the neck. The necrosing action of pus is no respecter of them. I prefer still to take the pharyngo-maxillary fossa along the simpler lines of my original description stressing the point that the tonsil which is attached to the superior constrictor (the inner boundary of the fossa) is the chief source of infection in the fossa. Also, it is worth remembering that there is a weak fascial boundary between the right and left pharyngeal fossae in the midline of the neck. In my opinion, the phrase "the carotid sheath is the Lincoln Highway for pus in the neck" still emphasizes a very practical point.

By lifting the submaxillary gland from its bed and turning it upward, the surgeon has a quick and simple approach to the base and the side of the tongue; to pus pocketing in the pharyngomaxillary fossa above, and to pus in or along the

carotid sheath below. It is a surgical revelation when one first tries the procedure on the cadaver. A finger in the fossa easily palpates the attachment of the tonsil as it lies on the inner surface of the superior constrictor. As infection of the pharyngo-maxillary fossa almost always originates in the tonsil, a necrotic pocket is often found opposite the tonsil. I once found such a pocket in a case of Ludwig's Angina. Even when pus is quickly found in the pharyngo-maxillary fossa I feel that it is worth while to explore with the finger the area of the attachment of the tonsil. If this is done the surgeon will get some illuminating surprises.

Of course, when the upper deck is densely infiltrated, exposing the submaxillary fossa is not so easy or so quick as when the operation is demonstrated on the cadaver, but it is still relatively simple.

When in deep abscess of the neck the maximum swelling is opposite the angle of the jaw most of the pus will be found above in the pharyngo-maxillary fossa; when the swelling is frankly on the side of the neck, it will be found either within the carotid sheath or running along its side.

The complications of deep infection of the neck are either thrombosis of the internal jugular vein with fatal sepsis or descent of pus along the carotid sheath, breaking into the trachea and producing a quick tragedy. This second happening I have never seen occur, but the first one occurs constantly. The general surgeon remains smugly unaware of what is going on. I have talked at him with my best language for 20 years, but to no avail. I have come to the conclusion that, with me at least, 20 years is a short time in which to put an idea over.

After a case of unilateral tonsillitis, even when the local throat signs have cleared up, if a septic temperature occurs, the jugular vein should be uncovered and inspected for thrombosis.

Dr. August L. Beck, who has had a large experience with neck infections and has written the best paper on the subject

that I know of, stated to me recently that chemotherapy, especially penicillin, will frequently cause a beginning neck infection to subside and completely resolve. Sometimes it takes all the altruism which one can muster to welcome the brushing aside of surgery by chemotherapy.

When I was in the medical school one of the surgical gods to us as students, was Dr. Cheever of the Boston City Hospital. He was tall, thin and looked every inch the part of the cool, collected surgeon. His words were short and to the point. He did not try to be a showman. He spoke with the assurance which comes from years of surgery. He did not litter up the lecture room with charts and models; to me he was a lonely figure giving us naked and vital surgery.

Dr. Cheever published a small book on surgery. I quote what he says about deep pus in the neck. To me his description is most striking. He almost hit upon thrombosis of the internal jugular vein associated with deep pus in the neck.

"Quite as rare and much more fatal is the deep-seated abscess of the neck, of which the hospitals see perhaps three to six cases a year, and a private physician, perhaps, not one in five or six years. It is a cellulitis followed by the formation of pus; acute, under the deep cervical fascia; usually somewhere above the sheath of the vessels near the sterno-mastoid muscle. Its symptoms are obscure. The patient has difficulty in swallowing, difficulty in breathing; the voice is affected, becomes hoarse. On looking into the mouth absolutely nothing is seen. The velum and palate and tonsils and the back of the pharynx, and probably all the parts below, are found free from irritation, except congestion of the vocal cords from pressure; but I am inclined to think the dyspnea produced is not due to pressure of the pus upon the parts about the larynx, because they are strongly protected by the box of the larynx, but by pressure on the inferior or superior laryngeal nerves. At the same time, from pressure on some fibres of the pneumogastric there is difficulty of swallowing, and imperfect action of the constrictors of the pharynx. The patient does not show anything inside of the throat; but

speedily there begins to show on one side of the neck a brawny, indurated swelling, but generally no redness. There is inability to move the neck, and edema, together with marked and severe constitutional symptoms; chill, high temperature, sweats. This means pus in deep, beneath the deep cervical fascia. If diagnosticated and reached, the patient is almost always saved; if not, they are generally fatal; and this by running down into the mediastinum and pleura, or breaking into the back of the trachea, or by suffocation. It is an extremely serious affection, and the whole point consists in making an early diagnosis; and, having made it, in pursuing the proper course, which is to seek for pus. We make the incision on the front or back of the sterno-mastoid muscle; the back is usually farther away from the pus. Then we take a director, and begin to bore carefully into the soft tissues of the neck; or we may get in the little finger. At first you may be quite disappointed. Finally, far up behind the styloid process, you get a gush of pus; enlarge the incision, insert a drainage-tube and wash out the whole cavity. Usually a free opening on one side, with drainage and syringing, suffices to relieve the trouble. The relief is wonderful, and recovery is prompt. They generally get well in about a fortnight after the incision is made."*

TYING THE EXTERNAL CAROTID.

In actual practice tying the external carotid artery to control nasal or tonsillar hemorrhage is not always so easy as it seems from the books. A neat way to find it is to pick up the hypoglossal nerve in the submaxillary fossa where it makes a triangle with the tips of the two bellies of the digrastric muscle (Lesser's Triangle) and follow the nerve outward and under the posterior belly of the digastric. Before the nerve hooks round the occipital artery it crosses the root of the external carotid. Make certain you have a branched artery and you are sure that you have the external carotid. The branch you usually find is the superior thyroid, the first branch of the external carotid.

*Lectures on Surgery—Cheever—pages 541-543, Damrell and Upham, Sec. Ed. 1898.

LUDWIG'S ANGINA.

Ludwig's Angina, I believe to be an abscess at the base of the tongue which has worked forward along the fascial planes of the muscles of the floor of the mouth. It can be reached by a midline incision through the under surface of the tongue but the base of the tongue must be reached to evacuate the main pocket of pus. The submaxillary approach makes the base of the tongue superficial and easily incised, and for this reason I prefer it. In one case I found the abscess pocket opposite the attachment of the tonsil in the pharyngomaxillary fossa.

When both sides of the neck are equally swollen, the surgeon is in doubt on which side to use the submaxillary approach. The patient may be able to tell on which side of the throat the pain started. Naturally in such a case the approach would be made on that side. Suppose a dry tap results from the first procedures, then it should be repeated on the other side. This may seem a bit formidable, but the surgeon is dealing with a life and death case, and incisions do not count. I prefer a generous T shaped incision for the submaxillary approach.

PERITONSILLAR ABSCESS.

I believe that a peritonsillar abscess is primarily an abscess of the soft palate. As the roof of the supratonsillar fossa is the under surface of the soft palate, this fossa offers the shortest route to the pus. In my experience pus can be reached by an incision through the supratonsillar fossa earlier than by the conventional incision through the root of the anterior pillar. The levator of the soft palate runs through the abscess and makes it a roughly bilobed pocket, the greater amount of pus being behind the muscle. In the approach to the abscess through the root of the anterior pillar unless the abscess is very large but little pus is obtained until the knife has pierced the levator muscle and the incision in the muscle has been dilated. This explains the dry tap which the operator often gets until the incision through the muscle has been stretched.

In opening a peritonsillar abscess one wonders at first where the internal carotid artery is located. Frozen sections of the head put it at least a quarter of an inch behind the posterior pillar of the tonsil. If the abscess contains a dram or two of pus, as it often does, this should make the artery at least three-quarters of an inch away from the point of the knife. Often, in opening a peritonsillar abscess the point of the knife has to be sunk almost half an inch and the incision spread before pus is obtained.

Except in recurrent attacks of peritonsillar abscess, one rarely gets pus before the third day. It has been my practice to make an exploratory incision at this time and if no pus is obtained to wait another three or four days. As the abscess customarily breaks without interference, a patient who has experienced this seldom has much patience with repeated fruitless punctures, for which you cannot blame him.

GLOBUS HYSTERICUS.

Cases of globus hystericus have disappeared from the Throat Clinic at the Infirmary. In the great majority of cases the symptoms are due to post-cricoid webs or webs in the pyriform sinus. Such webs are easily missed if the examination of the mouth of the esophagus is carried out with small tubes. With the larger esophagoscope they are readily seen and evulsed or bitten away with appropriate forceps. A short course of bouginage prevents the return of the web. From their position such webs cause fluid to overflow into the larynx, which produces a terrifying spasm. Patients with post-cervical webs live in terror of taking food, especially liquids. The relief of their symptoms by operation is most dramatic.

CERVICAL EXOSTOSES.

Cervical exostoses are frequently encountered in the orthopedic clinic and give no symptoms. In cases of difficulty in swallowing, however, as we see them at the Infirmary, cervical exostoses are often found associated with narrowing of

the esophagus opposite the exostoses. The exostoses occur most often on the bodies of the sixth and seventh cervical vertebrae. The circular narrowing of the esophagus caused by them has to be dilated with care for fear of too much trauma of the esophagus against the exostosis.

Before the passage of an esophagoscope, especially the larger tubes preferred by the writer, a lateral X-ray plate of the neck should be taken to rule out the presence of an exostosis. Years ago in the early days before I learned about cervical exostoses, I had a tragedy (gangrene of the upper end of the esophagus) by forcing too large a tube by an exostosis, the presence of which was unknown to me.

THE LATERAL X-RAY OF THE NECK.

There is another condition where the lateral X-ray of the neck gives valuable information. The prevertebral connective tissue holds the upper half of the esophagus loosely and responds very quickly to trauma or infection. Even after a routine and uneventful esophagoscopy, the X-ray will quickly show a slight thickening of the connective tissue behind the esophagus. This frightened me when I first observed it, but I soon found that it subsided quickly and without incident. In cases of the difficult removal of foreign bodies with trauma to the esophagus or perforation, the post-esophageal swelling is marked and can be used as a measure of the infection in the mediastinum, whether it increases in spite of chemotherapy and when drainage of the post-esophageal space is indicated.

PHARYNGEAL POUCH OF THE ESOPHAGUS.

The pig has two pharyngeal pouches. Embryologically man also has two, but both disappear. The fact that man has had pharyngeal pouches in the embryo suggests a possible embryological background for some of the pharyngeal pouches in man. A more important fact, however, is that in most cases of pharyngeal pouch in man the entrance to the esophagus is asymmetrical; it is off center and partially glued up.

X-ray plates show this clearly. When this formation of the opening of the esophagus is present the force accompanying the swallowing of food is off center and brings unequal pressure on the weak triangle of the upper end of the esophagus. It is through this triangle that the hernia of the esophageal pouch occurs.

I treated a small series of good sized pouches by slitting the upper half of the common wall between the esophagus and the pouch. It gave immediate symptomatic relief in five cases, but the last case and the one which I hoped would be the best of all, resulted in a tragedy. A mediastinal abscess occurred and the patient died of hemorrhage from the internal jugular vein; naturally I abandoned the operation. From this time on I turned these cases over to Dr. Lahey who did a two-stage external operation under local anesthesia. In the years which have passed since my unfortunate case he has operated over a hundred cases, some of the patients being well over 70, with only two deaths. I have never done the external operation, but I am glad to say that the surgeons at the Massachusetts Eye and Ear Infirmary are successfully doing it.

FIBROSIS OF THE TERMINAL PORTION OF THE ESOPHAGUS.

Early in my work on the esophagus (and this dates back more than 25 years) in the cases which came to the hospital labeled cardiospasm it was found that the fluoroscope very rarely showed spasm of the esophagus and the esophagoscope routinely disclosed a narrowing of the lower end. Anatomical studies located the point of narrowing at the upper edge of the left crus where it passes behind the esophagus and the esophagus bends to the left and becomes a narrow, flat ribbon as it passes through the crural canal to reach the stomach. Histological examination of autopsy specimens showed that fibrous tissue replaced the musculature of the lower end of the esophagus and that chronic infection of the gall bladder and of the liver was present in a majority of the specimens, and probably was the original source of the infestion in the esophagus.

The modification of the Sippey balloon bag by painting barium lines on it so that it could be seen by the fluoroscope and the amount of dilatation watched and the air pressure needed for dilatation controlled and recorded on a manometer, brought surgery by sight into the treatment of fibrosis of the lower end of the esophagus.

This was one of the chief landmarks of my surgical career. It was soon found that although the esophagus when seen by the fluoroscope and by the X-ray plate seemed absolutely closed below the point of fibrotic narrowing, it almost always would allow passage of the tip of the flexible finder with which the balloon bag was fitted. This finder measures 30 French. Putting all my findings together I now hold that the narrowing of the lower end of the esophagus formerly called cardiospasm is not spasm but a fibrotic narrowing due to infection; that the infection in the majority of cases has its origin in chronic infection of the gall bladder or the liver; that the ideal treatment is controlled dilatation by fluoroscopic sight, and that the proper name for the condition is fibrosis of the lower end of the esophagus.

After a certain degree of dilatation has been obtained by sight the mercury bougie can be safely substituted for the balloon bag. This can be passed by touch, although I prefer to pass it under the guidance of the fluoroscope for a while. Finally the patient can pass a mercury bougie of appropriate size on himself and so lengthen the time between the visits to his physician. Taking a hint from the mercury bougie, the whalebone staff of the instrument which I have been accustomed to use could with advantage have the whalebone staff done away with, and the shaft of the instrument replaced by a rubber tube filled with mercury like the mercury bougie.

To my mind one of the most important advances in the etiology of narrowing of the lower end of the esophagus was made by a South American investigator who found a food deficiency analagous to beri beri to be the cause of a series of cases of dilated esophagus with obstruction at the lower end giving the X-ray picture of advanced cases of fibrosis due to infection.

CANCER OF THE ESOPHAGUS.

All my life I have stood helpless before cancer of the esophagus. I have tried all the conventional methods of treatment without success. At one time it seemed as if X-ray could accomplish something. Cases were reported where the tumor disappeared under X-ray treatment, but it left the esophagus thinned and fibrosed, so that when the patient was given solid food the esophagus ruptured and death resulted.

Today thoracic surgery has brought great hope to patients with cancer of the esophagus. By the thoracic approach cancer of the esophagus can be reached almost anywhere in the chest. Dr. William Sweet of the Massachusetts General removed a cancer near the middle of the thoracic esophagus and demonstrated that the stomach could be brought up for anastomosis even to the arch of the aorta. Some of his cases are two or three years old and give great promise of full success.

Speaking of the thoracic approach to the esophagus, it can be used not only for cancer of the esophagus but also for strictures which cannot be dealt with by dilatation. This again is an epochal advance in surgery of the esophagus.

THE PLUMMER VINCENT SYNDROME.

In the few cases of the Plummer Vincent syndrome which have been referred to me from the medical department of Massachusetts General Hospital, I have always found a history suggesting stricture of the upper end of the esophagus and on esophageal examination have found such a stricture. So far I have felt that the cart has been put before the horse. I firmly believe, however, that in some cases vitamin deficiency may be the cause of the condition of the tongue and be back of the difficulty in swallowing.

BRAIN ABSCESS.

During the first World War when I was serving with the First Harvard Unit in France under the British, the English

surgeons were draining deep brain wounds and brain abscess with zinc tubes pierced with multiple perforations. On returning home I devised the wire gauze brain drain. The idea of the wire gauze was that each hole in the gauze was a possible drainage channel. The gauze drain was supposed to be kept clean and so draining by daily curetting of the granulations which forced their way through its meshes. The drain was made of various sizes for various sized cavities. This meant that a certain stock of the drains had to be kept on hand. This proved to be a drawback outside of our hospital. It was much easier to take a piece of rubber tubing, cut it the proper length, and use it as a drain. Naturally, as the wire gauze drain was, so as to speak, my child, it was used almost routinely at the Infirmary during my service there.

The drain was left in for three weeks. At the end of this time it was found that in some cases connective tissue held it so firmly that it had to be dissected out, sometimes under anesthesia.

In connection with the use of the wire-gauze drain, Dr. Fred, of the Infirmary, made a significant observation in one of his cases: after the drain had been in three or four days, he removed it in order to investigate the abscess cavity, and to his surprise, found that the abscess had collapsed, so he did not replace the drain. Because of this observation I have wondered if it would not be well to remove the drain of any type on the third or fourth day to see if the fortunate happening which Dr. Fred had in his case would not repeat itself.

To me, a thoroughly surgical way to treat a brain abscess is as follows: Suppose the surgeon is dealing with a temporosphenoidal abscess. After a sufficient amount of bone has been removed in the typical place over the temporal bone, the dura is walled off with the galvano cautery by drawing a line about the edges of the bone opening. The brain is then explored by inserting the Cushing needle through the dura, and the pus is found. With the needle held in place as a guide, a cone of brain tissue is excised with the cautery knife down

through the overlying brain tissue and through the capsule of the abscess. This gives an opening into the abscess which satisfies one's surgical instinct. If a wire gauze drain is available and is preferred, it is inserted and held in place by one suture through the skin. If it is not handy a rubber tube drain is used. At the end of three or four days, the gauze drain is removed to see if the abscess tends to collapse. If it does the drain is left out. If the drain is replaced, at the end of a week it can be removed again. If the inner wall of the abscess seems to be coming to the surface, the drain is left out, and the abscess capsule may be left in place to "wither on the vine," so to speak, or cut off. If it can be left in place or thinned down it will offer a certain protection against hernia of cerebral tissue. One recognizes in this procedure the practice and teaching of King.

In the early days of my practice of otology, the custom was to approach a temporo-sphenoidal abscess through the stalk of the abscess when this drained through the roof of the middle ear. It was held that the stalk was walled off and so offered a safe approach. It was a blind route and to that extent unsurgical. Often it was not realized at first how large the abscess was. This method was not followed for long as the otologist soon saw the light and adopted the temporal route.

With this bit of otological surgical history out of the way I should like to discuss briefly the chief present day methods of dealing with a brain abscess. There are two competing techniques, — the open method and the method which relies on multiple trocar punctures for drainage. I have just given in detail the method of open drainage which appeals to me. King follows the open method and relies on the abscess coming to the surface where it can be excised in whole or in part, as the circumstances of the case dictate. The method of drainage by repeated trocar punctures is a favorite of the neurosurgeons. The basic idea, if I understand it right, is to drain the abscess by repeated trocar punctures until it remains dry and then let it obliterate by connective tissue and remain as an innocent foreign body in the cerebral tissue. This method

rubs my surgical sense the wrong way. It seems to me like depending upon a straw to save a drowning man. Every trocar puncture offers a new chance to infect the meninges. It is a supreme example of blind surgery.

Results, however, are what count in any method of treatment. I confess that our results at the Infirmary with the open method so far have been poor. The neuro-surgeons at the Massachusetts General Hospital maintain that their results are better than ours at the Infirmary. If they prove to be right there is nothing for the advocates of the open operation to do but to improve their type of operation or turn to the trocar method.

The neuro-surgeons are especially fond of the trocar method of drainage in what they call unencapsulated abscesses. How they can tell by feeling with the end of a trocar cannula the absence of a capsule is a sureness of touch which is beyond me. Dr. Mixter, formerly Chief of the Neuro-Surgical Service at the Massachusetts General Hospital, in a recent discussion of this subject with Dr. Schall said that the procedure is unsurgical but that it works; that is, the cases so treated get well. I can understand finding a brain abscess through a small trephine opening and then draining it once by the trocar cannula. This done, I feel that the size and position of the abscess should be determined by filling it with lipiodol or thorotrast. I cannot help feeling that an abscess of any size has a sufficient capsule to call for open surgery. Now that we have chemotherapy it is going to be harder than ever to evaluate any form of surgical technique which may be employed in treating a brain abscess, that is, how much credit to give to surgery and how much to chemotherapy. Any double procedure makes judgment difficult.

At the Harvard Club about two years ago (Nov. 8, 1944) I had a chance to talk about the treatment of brain abscess with Dr. Donald Munro, Chief of Service in the Department of Neuro-Surgery at the Boston City Hospital. I had been hoping to do this for a long time. I was glad to find that drainage of a brain abscess by multiple trocar punctures did not appeal

to him; further, he felt that a multilocular abscess could not be drained satisfactorily in this way. He added, "this was Dr. Cushing's method and his results were poor." Dr. Munro favors the open method. His procedure is: having found pus with the trocar needle he locates the abscess capsule with his finger, and then takes out a core of brain tissue with the cautery knife and then by sight removes a portion of the capsule, also with the cautery knife.

For years he has been using the wire gauze drain, and still favors it. He liked the idea, first used by Dr. Fred, of making a trial removal of the drain at the end of a few days, in order to see if the abscess has a tendency to collapse.

A new procedure, at least new to me, was lately reported at an Infirmary Clinical meeting by one of the newcomers to the Massachusetts General Hospital neurological service, Dr. Richard Sweet. His procedure is to find and evacuate the abscess, and if it is encapsulated, then immediately to dissect out the capsule. This done, he puts in a light drain and sutures the dura in order to prevent the old nightmare of a hernia of the brain. Three of his cases so operated were successful; in two, the patient died. The procedure is the acme of logical surgery.

In treating a brain abscess, if too much of the skull is removed there is danger of an increasing brain hernia which can defeat the whole operative procedure. This has happened to me a few times. It can be guarded against to a great extent by exploring for the abscess through a moderate trephine opening, and having found it by determining its size by thorotrast and then enlarging the bone opening to fit the abscess, or by making a second opening in a position to give the best drainage.

In an extensive extradural abscess one could make two moderate openings some distance apart. This is not necessary as a rule, because it is not until the dura is perforated that the danger of a brain abscess occurs. When a subdural abscess is present, at times this often perforates the dura; at other times the surgeon has to do it to reach the abscess. In either

case the danger of a brain hernia is in proportion to the size of the skull opening.

At the mortality meeting of the department of otolaryngology of the Infirmary held on Wednesday, Aug. 28, 1946, two fatal brain cases were reported. One was a case of tubercular meningitis in a five-year-old child and the other was a temporal lobe abscess in an adult who had respiratory failure after the evacuation of the abscess. The patient was revived temporarily but died soon after being returned to the ward.

In this second case there was a compression ring about the brain stem at the foramen magnum and another one which followed the edge of the tentorium on the side of the abscess. Dr. Kubik from the pathological department of the Massachusetts General Hospital, who demonstrated the autopsy specimens, said that the edges of the tentorium often gave a compression ring. This was new to me; so was his answer to a question of mine asking which was the more frequent as a cause of a compression ring about the brain stem, a temporal lobe abscess or an abscess of the cerebelllum. I had always supposed that a compression ring from abscess of the cerebellum was the great cause of respiratory failure in operations for brain abscess. Dr. Kubik answered that the pressure ring occurred about an equal number of times for each type of abscess.

As I look back upon my surgical training I have been surprised now and then how some elementary point escaped me for a long time. It was another case of being blind to the obvious. An instance of this is the following: it makes no difference whether the motor centers about the Fissure of Rolando — I was brought up on the older anatomical nomenclature — are pressed upon from within the brain or from without. That is, a brain abscess and an extradural abscess both can cause motor paralysis equally well. I know of one instance where a parietal extradural abscess was as large as the palm of the hand and naturally was accompanied by extensive paralysis. From my earliest surgical days, I at once thought of some intra-cranial condition as one cause of a motor paralysis but

it was many years before I sensed that an extradural abscess could do the same thing. As Sherlock Holmes would say, "Elementary, my dear Watson, elementary."

THE REMOVAL OF TONSILS AND ADENOIDS WITH THE PATIENT IN THE UPRIGHT POSITION.

Dr. Franklin Hooper, the first professor of laryngology at the Harvard Medical School, imported the adenoid operation into this country by way of Boston. The removal of tonsils, of course, had been done for years. For this operation the patient was held in a sitting position. The same position was used when adenoids were removed alone or by the combined operation; that is, removing both the adenoid and the tonsils at the same operation. This was the routine position well into my day. Outside of New England many operators considered the upright position dangerous and had the patient recumbent on the operating table in the Rose position. From time to time a lung abscess would follow tonsillectomy in our hospital, or cases where this complication followed tonsillectomy would be sent in from the outside.

When I was about half way along in my service as chief of staff, that is, after about seven years, I became suspicious of the upright position as a possible or a contributing cause of lung abscess after tonsillectomy. It happened that there were at the same time on the medical service of the hospital, five or six cases of lung abscess following tonsillectomy. Two were our cases, the others came from outside. All had been operated upon in the upright position. I then changed the operating position for the removal of tonsils and adenoids to the Rose position, and it has been continued to date. For years now, lung abscess after tonsillectomy has been very rare. It is hard, of course, to prove that the change from the upright position to the Rose position is the sole cause of the practical disappearance of this complication of tonsillectomy with us. As in most hospitals, the tonsil and adenoid operations are done by the senior House Officers. Many of the members of the staff, especially the older members, still use the upright

position in operating upon their private cases and have never had trouble enough to give it up.

THYMIC DEATHS.

In the November, 1924, issue of THE LARYNGOSCOPE, I published the following preliminary notice: "Following a thymic death a year ago of a child who had been etherized and was about to have his tonsils and adenoids removed, it was made a routine at the throat department of the Massachusetts General Hospital and the Massachusetts Eye and Ear Infirmary, to X-ray the chests of all patients from one to 16 years (the age limit is now one to 10 years), who were scheduled for the tonsil and adenoid operation. All children showing a broad superior mediastinum were considered suspicious thymus cases, and were given four X-ray treatments of a third of an erythema dose at intervals of 10 days."

This procedure was adopted by some of the local hospitals, notably the Boston City Hospital, and has been continued to date; likewise it has been continued to date at the Infirmary. Locally, in some hospitals it was met with derision and much sarcasm, and the contention which I held, that a child in whom the X-ray showed an enlarged thymus was a potentially tragedy case, was laughed off. A few years ago my successor as chief of service at the Infirmary, Dr. Schall, devoted a meeting to a review of the subject. At this I opened and closed the discussion. Both Dr. Schall and I, so to speak, were put on trial. I reiterated my position that a child with an enlarged thymus was a potential tragedy case and until someone could give me a better and easier way of picking out these potential tragedy cases I strongly advised continuing the preoperative X-ray on all cases which were to have the tonsil and adenoid operation. Dr. Schall agreed with me and the procedure is still in daily use at the Infirmary.

At this meeting one of our city medical examiners brought up the very practical point, which I had stressed years earlier, that if a lawsuit should follow a death after a tonsillectomy and no preoperative X-ray had been taken and a

malpractice suit was started, the attorney prosecuting the case could make this a strong point toward proving neglect.

None of the pathologists who took part in the discussion could answer my question as to what was the function of the thymus, in spite of all the new knowledge of the endocrine glands which has been accumulated in the past 15 years. Under the date of Nov. 13, 1944, Dr. Schall received a letter from the pathologist and medical director of the Children's Hospital at Denver saying that his attention had just been called to this discussion and the review of the stand taken at the Infirmary. He suggested that the adrenal gland might play a part in these so-called thymic deaths. This suggestion appeals to me somewhat. In lighter vein and to end what I have to say on this subject, and in spite of the rather poor taste of injecting a lighter note in a discussion of a subject so filled with tragedy, I might remind the reader that I said in the discussion of another subject, that it often took 20 years to put any of my ideas over, and it is just 20 years (1924-1944) between my first article and the adoption of the preoperative X-ray in tonsil and adenoid cases by the Denver Hospital. The above I consider one of my real milestones.

THE OPEN TRACHEOTOMY.

By open tracheotomy I mean that instead of the usual slit in the trachea, the actual removal of a circular piece of the front wall, and not inserting a tracheotomy tube. I have spoken of this procedure at various meetings a number of times in discussions but have never directly reported it; however, a few years ago Dr. Hill of Waterville, Me., reported it fully and with due credit. Its advantage is that a tracheotomy case so treated does not have to go through the hazardous first night with its frequent complications. As Dr. Hill put it, if the surgeon does a tracheotomy in a distant hospital, he can with a clear conscience safely leave the patient under the care of the family physician who called him in. The important point in the technique of the operation is to dislocate the isthmus of the thyroid downward, cut it and suture the edge of each stump of the isthmus carefully so that all bleeding is

securely and permanently controlled. In order to keep the front face of the trachea free the halves of the isthmus of the thyroid are stitched firmly outward to the skin. The face of the trachea should be well exposed and the field dry before the patient is returned to bed. If at the end of a week it is necessary to keep the tracheal opening a cannula may be inserted as by this time the strap muscles of the front of the neck will have narrowed the field to a slit.

Without a tracheal cannula in place it is easy to insert a rubber catheter into the trachea if it plugs up. This eliminates the danger of slipping the tracheal cannula between the facial planes of the neck, an accident which often happens in unskilled hands and even in skilled hands, with subsequent alarming emphysema of the neck. If tracheotomy becomes necessary after a peanut case and the lungs fill up, as happened in a case of mine, they can be drained, especially if the patient is a child, by holding it up by the heels.

A theoretical objection to the removal of a section of the front wall of the trachea is the possible occurrence of stricture. No such happening has been reported following the operation, and the first operations of this kind were done at least 15 years ago.

The routine use of a small bronchoscope in the trachea or the writer's "life saver" converts an emergency tracheotomy into a quite deliberate one.

INSTRUMENTS.

It is exciting to dream up a new instrument and then proceed to have it made. It is an expensive pleasure, and you never get your money back. I must have devised 10 or a dozen instruments. Those that have had a survival value are the long and short oval esophagoscope, the open adjustable laryngeal speculum, an applicator for direct intubation, a spiral wire papilloma remover for the larynx, the safety pin closer, the modification of the Sippey balloon bag for fluoroscopic dilatation of fibrosis of the lower end of the esophagus.

When you invent an instrument you should remember that

it was devised to meet your own needs and to fit your own hand, and that it may not fit the hand of the next surgeon.

In using any form of safety pin closer the surgeon should practice with it on a duplicate pin outside of the body before trying it on the living.

The subject of designing instruments puts me in a reminiscent mood. While I was in the Boston Latin School, and used to haunt the second hand book shops on Cornhill, I often went 'round the corner to see the window display of surgical apparatus and instruments in the shop of Codman and Shurtleff, then located on Tremont Street near Adams Square. The display was of the old fashioned, motley type. Artificial arms and legs, crutches, flat foot plates and trusses of weird form were the staple articles. For some unknown reason the window had a peculiar fascination for me. I have wondered since when in an egotistical mood, whether its attraction was not the foetal stirring of my mechanical bent. My little flair for inventing came from my father who was something of an inventor.

Codman and Shurtleff at the time I discovered their show window were the chief surgical instrument makers and dealers in Boston and New England. Their stamp on an instrument insured that it was of the highest quality, and the price also generally was high. One mentioned an instrument of theirs with the same pride that one spoke of an instrument made in Paris by Luer.

When I started in practice I soon began to know the Codman and Shurtleff shop from the inside, not merely from its window display. They had moved by this time to the lower half of Massachusetts Avenue, half way to Symphony Hall. They followed the business trend up town, and followed the doctors also. The only doctor left on Beacon Hill was Dr. J. Collins Warren (Col Warren to those of us who were his House Officers). He was known for his Bunker Hill ancestry, and for his radical operation on the female breast. To get back to Codman and Shurtleff; it was in their new location that I first met their outdoor salesman, Mr. John Given. He made reg-

ular rounds of the hospitals and secured the best trade in the city. For years it would have been considered a sin for the Massachusetts General Hospital not to place their instrument orders through him.

He lived in Scituate, Mass., on the South Shore, and was an ardent sailor. Every year he brought his boat for race week to Marblehead Harbor. It was a small, one-man boat, but I forget its exact type. He lived alone on it during this week, and for this once in his life forgot surgical instruments. As I had my summer home on the old town side of the harbor he always paid me a rather ceremonious visit. I remember his very jaunty cap with the insignia of his local yacht club. Never having owned a boat of any kind nor possessed a yacht club cap, although I had long been a member of one Marblehead yacht club, I was very much impressed by his trim appearance and felt that I had missed my opportunity. He was skipper and crew of his boat. He was no longer a salesman; he was the captain of his ship and of his soul.

What I am leading up to is this: When I began inventing instruments I would make a rough model of my idea and hand it over to Given. He saw it through the Codman and Shurtleff machine shop and then submitted it to me. Sometimes he suggested worth while improvements. Generally, especially with my safety pin closer, four or five models were submitted and worked over before the instrument was finally perfected. My association with Mr. Given lasted for 15 or 20 years. It was a very pleasant comradeship, and a very profitable one to me; I am quite sure that it was profitable also to his firm.

I never had a machine shop of my own as Yankauer did. He made most of his instruments and invented not a few, so I missed the pleasure and the satisfaction of making my own instruments. Quite a few surgeons, however, have this accomplishment. I have always envied them.

PRECEPTS.

The following are favorite precepts with me:

Operate by sight whenever possible.

Take the shortest route.

Timid incisions breed timid surgery.

Small incisions give a small operating field, especially deep in the neck.

When in doubt about infection of the mastoid, do an exploratory operation. You will satisfy your surgical conscience and get many surprises. An X-ray cannot be relied upon 100 per cent.

In making a diagnosis think of the common happening first. Mistrust a fancy diagnosis. The common is what commonly happens.

127 Front Street.

THE EFFECT OF SMOKING ON THE CALIBER OF THE SUPERFICIAL BLOOD VESSELS IN THE UVULA AND THE SOFT PALATE.

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INTRODUCTION.

Numerous observers have reported from time to time on the possible irritating effects of cigarette smoke on the respiratory system. Ballenger[1] and Flinn[2] observed color changes in the throat and obtained polemic results. The former found no difference in irritation among three types of special cigarettes (glycerine treated, diethylene glycol treated and cigarettes containing no humectant), and the latter found reduced irritation following the smoking of cigarettes moistened with diethylene glycol as compared with those moistened with glycerine. Myerson[3] reported a greater increase in the diameter of the blood vessels of the uvula following the smoking of commercial brands of cigarettes presumably treated with glycerine than of a commercial brand of diethylene glycol treated cigarettes and interpreted this to mean that a corresponding difference in irritant action existed. As all of these investigators employed methods in which the judgment of the observer was the determining factor, it seemed desirable to attempt to make comparisons of various types of cigarettes employing a technique not dependent upon observation or judgment, but which allows exact measurement of the change as recorded on photographs of the area involved at the time of the reaction.

The structures observed by Myerson appeared to be susceptible to such measurement and, therefore, this investigation was directed toward the examination of the effect of

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smoking on the blood vessels of the uvula and soft palate; however, it seems that this was not the optimum field for observation since these structures are constantly in motion and are expanding and contracting. Consequently it is difficult to determine whether changes are the result of this motion or are due to external stimuli. It is probable that the hard palate would have been a better field for study, but in view of Myerson's work the field he described was used.

The method employed in this investigation in brief comprised photographing the uvula and soft palate of a subject immediately before and after smoking a test cigarette and measuring the blood vessels on the film.

APPARATUS.

The apparatus employed consisted of a rebuilt Cameron surgical camera using the two-inch focal length lens furnished with the camera, and Wabash "Press 40" flash bulbs. The fixed focal point of the lens was at a distance of six inches from the lens. The camera was modified by installing a focusing light of considerably greater candlepower than that furnished with the camera; use of a lens stop of f:18; removal of the neutral density filter from the inside of the camera; replacement of the ground glass focusing screen with a clear glass focusing screen in order that focusing by the parallax method could be employed; and the addition of a device designed to maintain a fixed distance between the camera and the upper incisors of the subject. This last device was of great importance since the size of the image is inversely proportional to the distance from the lens to the subject. Consequently, in order for the final measurements to be of any value it was necessary that the distance be the same for all photographs in a given series or an individual subject. The apparatus is shown in the accompanying photographs.

The film used in the camera was Type A Kodachrome. Prior to its adoption considerable experimentation was conducted on other types of film, including infrared with the

proper filters and panchromatic with Wratten C-5 (blue) and Wratten B (green) filters. The photographs obtained on infrared film showed no vascular pattern, and it is possible that these vessels carry blood of a rather high oxygen content since it is well known that venous blood is opaque to infrared light. The use of panchromatic film with the Wratten filters resulted in superficially good definition of the vascular sys-

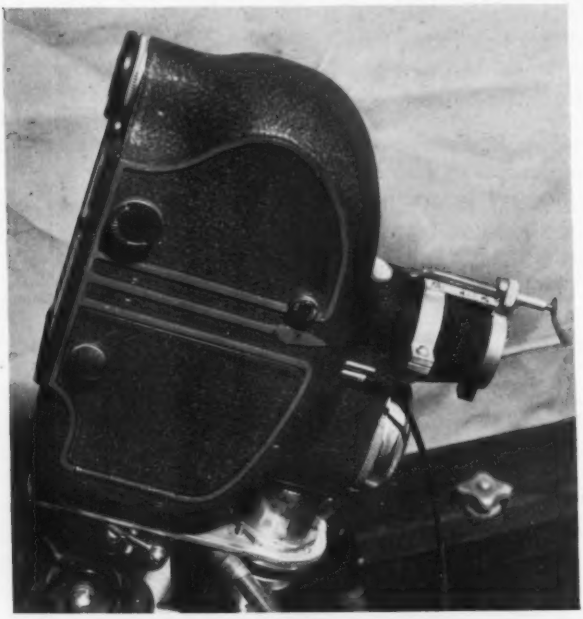

Fig. 1.

tem, but it was found that the grain of the film was too coarse to permit the great enlargement necessary for convenient measurement of the vessels and so the use of Kodachrome was adopted. The method used involved projecting the images on a ruled screen under carefully controlled conditions and measuring with a millimeter scale those of the vessels which

were sufficiently well defined. The maximum error of the readings is estimated at ± one-half unit of the scale. The rulings on the screen were used as coordinates to identify the vessels measured. All measurements were made by the same person (L. H. J.).

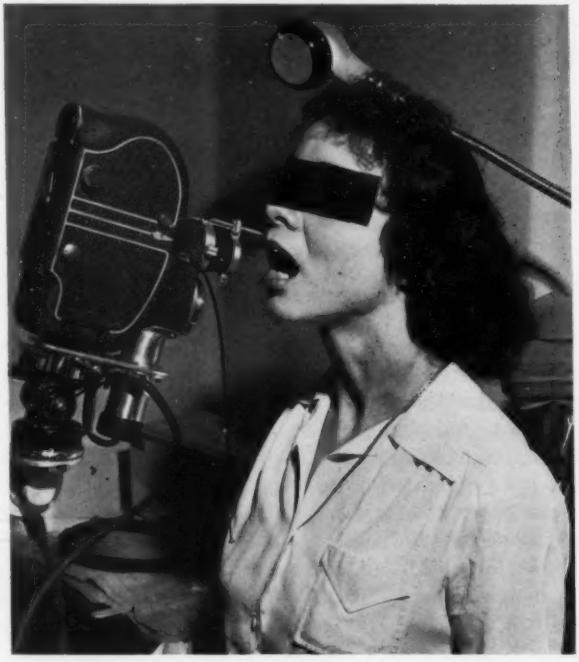

Fig. 2.

PROCEDURE.

Each subject had been instructed to forego eating, drinking and smoking for at least an hour before the test. On reporting, he was put through the following routine: An examination of the subject's throat was made and a measurement was then taken of the distance from the upper incisors

to the tip of the uvula when the throat was completely relaxed and the appropriate setting made on the positioning device of the camera. All photographs were made under the same conditions of throat relaxation. A photograph of the legend on the data sheet including the number of the subject, the number of the film and the date was then taken. A photograph of the subject's uvula and soft palate was then made and the subject instructed to smoke in his normal manner 10 puffs on a cigarette furnished him. Immediately following the tenth puff a second photograph of the subject's uvula was taken. The subject was allowed to rest without smoking or drinking for at least 10 minutes and a third photograph was taken of his uvula. A second cigarette was then given him and he was instructed to smoke in the same manner as with the first cigarette. Immediately following the tenth puff on the second cigarette a fourth photograph was taken. In series UA involving the use of three cigarettes on each subject, a fifth and sixth photograph were taken after a second 10 minute rest period.

It was necessary to use a tongue depressor with practically all of the subjects since few of them had sufficient control of their tongues and soft palates to furnish a clear field. The tongue depressors used varied somewhat, although the same one was always used for the same subject. Metal depressors were used on most of the subjects, but in a few instances it was found necessary to use wooden ones, due to slipping of the metal. Some difficulty was experienced with a small proportion of the subjects in that the use of a tongue depressor resulted in gagging. This was usually ascertained during the preliminary examination and in all instances where gagging occurred the subject was dismissed and no data recorded.

The cigarettes used in the test had all been stored in a humidity chamber over saturated calcium nitrate solution at 50 to 55 per cent relative humidity. The cigarettes were rotated with each subject so that on the average with the special cigarettes one-third of the group of subjects started with each of the three types of cigarettes, and with the branded cigarettes one-half of the subjects started with the

control brand, and the balance with one of each of the four other brands.

The investigation was divided into three series of tests. Series UA covers the tests on special cigarettes which had been prepared under commercial conditions to contain: *1.* 3.65 per cent of glycerine, *2.* 2.74 per cent diethylene glycol, and *3.* no humectant. Series UB consisted in comparing one brand designated as the control brand (A) with each of four other brands (B), (C), (D) and (E). Series UC consisted of a group of photographs taken of the uvula of 12 non-smokers. The second photograph in this case was taken five minutes after the first photograph; the subjects, of course, did not smoke during the test.

SERIES UA – SPECIAL CIGARETTES.

The cigarettes used in this series had been treated during their manufacture with either 3.65 per cent glycerine, 2.74 percent diethylene glycol or no humectant. These cigarettes were coded and the code was unknown to all except one of us (C. W. L.) at the time the work was conducted. The concentrations of humectant employed were those reported by Mulinos and Osborne[4] to approximate normal commercial practice. The cigarettes were made from three 2,585 pound batches of tobacco with the regular production equipment of one of the large cigarette manufacturers in the presence of one of us (C. W. L.).

This series consists of the examination of 12 subjects. The uncut strips of film were submitted for measurement, accompanied by a blank data sheet. In order to avoid any possible psychological factors, there was no indication on these sheets after the first few subjects as to the types of cigarettes employed or whether the subject had smoked at all.

There are several different possible methods of analyzing the data as follows:

1. The average response of all the subjects to the three types of cigarettes was calculated by determining the mean

diameters of all the vessels apparent in the photographs for each subject before and after smoking and from these data calculating the percent change for each subject; the average of the percent change for all subjects represents their average response.

Glycerine treated cigarettes.....2.8% *decrease* in size of vessels
Diethylene glycol treated cigarettes.....6.3% increase in size of vessels
Cigarettes containing no humectant.....1.3% *decrease* in size of vessels

2. The average response of all the blood vessels to the three types of cigarettes was determined by calculating the per cent changes in the diameter of each vessel apparent in the photographs following smoking and from these data calculating the mean.

Glycerine treated cigarettes.....3.1% increase in size of vessels
Diethylene glycol treated cigarettes.....5.4% increase in size of vessels
Cigarettes containing no humectant.....1.3% increase in size of vessels

3. The response of an average subject was determined by calculating the mean diameters of all the vessels apparent in the photographs for each subject before and after smoking, obtaining the average diameters for all subjects and from the differences between the averages calculating the per cent change.

Glycerine treated cigarettes.....3.2% *decrease* in size of vessels
Diethylene glycol treated cigarettes.....5.4% increase in size of vessels
Cigarettes containing no humectant.....3.5% *decrease* in size of vessels

4. The response of an average blood vessel was determined by calculating the mean diameter of all the vessels apparent in the photographs before and after smoking and from these means calculating the per cent change.

Glycerine treated cigarettes.....1.1% increase in size of vessels
Diethylene glycol treated cigarettes.....3.7% increase in size of vessels
Cigarettes containing no humectant.....No change in size of vessels

5. In addition to the above quantitative data it is possible to obtain qualitative information by counting the number of increases, decreases and no changes observed. When this is done for the individual blood vessels the following results are obtained:

	No. Increased	No. Decreased	No. Showing No Change
Glycerine treated cigarettes	10	9	16
Diethylene glycol treated cigarettes	15	10	14
Cigarettes containing no humectant	5	5	21
All	30	24	51

Thus of all the increases observed, 50 per cent followed the smoking of diethylene glycol treated cigarettes, 33 per cent followed the smoking of glycerine treated cigarettes, and 17 per cent followed the smoking of cigarettes containing no humectant.

6. Similarly, the counts of the number of subjects showing average changes result in the following data:

	No. Increased	No. Decreased	No. Showing No Change
Glycerine treated cigarettes	3	4	4
Diethylene glycol treated cigarettes	6	4	1
Cigarettes containing no humectant	3	5	2
All	12	13	7

As before, of all the subjects showing an average increased diameter, 50 per cent were again the results of smoking diethylene glycol treated cigarettes, 25 per cent from glycerine treated cigarettes, and 25 per cent from cigarettes containing no humectant.

On the basis of all the above data one is forced to conclude that the diethylene glycol treated cigarettes used in this investigation may have had a greater dilating effect on the blood vessels than either the glycerine treated cigarettes or the cigarettes containing no humectant. The differences observed in the quantitative data (paragraphs 1-4) are so small as to cast doubt on their significance; consequently, a statistical analysis was made of the data covering the average response of all of the blood vessels (2, above) and the subject averages (3, above). It was found that neither the differences observed between the average response of the vessels nor the differences in average reaction of the subjects are significant; consequently, while the sample showed a slightly greater effect on the 105 measurable blood vessels of the 12 subjects as a result of smoking diethylene glycol treated cigarettes as compared with glycerine treated ciga-

rettes and cigarettes containing no humectant, it is questionable whether or not this difference would necessarily hold true for other samples.

SERIES UB – BRANDED CIGARETTES.

The cigarettes used in this series were the five leading brands. In an experiment of this sort involving five treatments the choice must be made between the various experimental designs possible. One design might consist of having each subject smoke only one brand of cigarettes. The results for each of the five groups of subjects could then be compared. With a large enough number of subjects in each group, the desired information could be obtained.

A second design might be similar to that used in Series UA in which each subject smoked all of the types of cigarettes under test; however, with five cigarettes the time required for each subject would be impracticably long.

A third design, the one which was used in this experiment, involved selecting one brand as a control which all subjects smoked and dividing the other four brands among the subjects so that one-fourth of the subjects smoked one of the brands other than the control. In this manner it is possible to secure information on the variation between subjects as a result of smoking the control, as well as the possible differences between the control and the other brands.

In this experiment 112 pairs of tests were made in which all subjects smoked the control cigarette (A). Twenty-eight tests on each of the four other brands were made. While 112 pairs of smokes were made, only 97 subjects were used, as 13 were used twice and one was used three times. Hence, it was possible to determine the constancy of a subject's reaction to the control brand by comparing the data for those subjects on which duplicate tests were made.

As in Series UA, there are several different methods of analyzing the data obtained by measuring the photographs of

the blood vessels. The data show by the same methods of calculation described for Series UA the following results:

1. The average response of all the subjects to the test cigarettes:

B vs. A— B, 0.3% increase; A, 9.9% increase in size of vessels.
C vs. A— C, 1.2% increase; A, 5.1% increase in size of vessels.
D vs. A— D, 6.4% increase; A, 0.2% *decrease* in size of vessels.
E vs. A— E, 2.0% increase; A, 4.7% increase in size of vessels.
All vs. A— All, 2.4% increase; A, 4.5% increase in size of vessels.

2. The average response of all the blood vessels to the test cigarettes:

B vs. A— B, 3.1% increase; A, 7.4% increase in size of vessels.
C vs. A— C, 0.6% increase; A, 4.0% increase in size of vessels.
D vs. A— D, 9.0% increase; A, 0.3% *decrease* in size of vessels.
E vs. A— E, 6.4% increase; A, 5.4% increase in size of vessels.
All vs. A— All, 4.4% increase; A, 3.7% increase in size of vessels.

3. The response of an average subject to the test cigarette:

B vs. A— B, 0.8% increase; A, 0.9% increase in size of vessels.
C vs. A— C, 0.9% increase; A, 4.6% increase in size of vessels.
D vs. A— D, 5.6% increase; A, 0.5% *decrease* in size of vessels.
E vs. A— E, 2.5% increase; A, 4.8% *decrease* in size of vessels.
All vs. A— All, 1.9% increase; A, 4.0% increase in size of vessels.

4. The response of an average blood vessel to the test cigarette:

B vs. A— B, 0.9% increase; A, 5.6% increase in size of vessel.
C vs. A— C, 0.3% *decrease*; A, 3.0% increase in size of vessel.
D vs. A— D, 7.0% increase; A, 1.6% increase in size of vessel.
E vs. A— E, 4.8% increase; A, 5.3% increase in size of vessel.
All vs. A— All, 2.7% increase; A, 2.7% increase in size of vessel.

5. The number of blood vessels showing changes:

	OTHER BRANDS			CONTROL (A)		
	No. Increased	No. Decreased	No. Showing No Change	No. Increased	No. Decreased	No. Showing No Change
B	21	18	34	18	7	20
C	13	12	51	17	11	47
D	21	4	40	16	21	34
E	18	8	23	16	10	24
All	73	42	148	67	49	125

Of all the increases observed, 48 per cent followed the smoking of the control cigarettes, and the balance of the increases was distributed among the other four brands. The

Chi-square test shows that the difference in the above counts (73 and 67) is probably not significant.

6. The number of subjects showing average changes:

	OTHER BRANDS			CONTROL (A)		
	No. Increased	No. Decreased	No. Showing No Change	No. Increased	No. Decreased	No. Showing No Change
B	6	6	6	7	3	4
C	7	5	7	10	5	5
D	10	2	5	5	6	9
E	7	3	4	5	0	8
All	30	16	22	27	14	26

Of all the increases observed, 47 per cent again followed the smoking of the control cigarettes, and the balance of the increases was distributed among the other four brands. The Chi-square test shows that the difference in the above counts (30 and 27) is probably not significant.

On the basis of all the above data obtained from the 504 measurable blood vessels of the 112 subjects, it is concluded there is no substantial difference between the effects of smoking the control cigarettes and the other four brands taken as a group. There are, however, fairly consistent differences between the control and three of the other brands. Brands B and C produced a smaller increase by each of the above analyses than the control, and on the other hand, the control effected a smaller increase than Brand D by each method of analysis. The score between Brand E and the control is about even by the six methods.

SERIES UC – TESTS WITHOUT SMOKING.

Since there are many uncontrolled variables in employing a technique of the sort used here on human subjects, it seemed worthwhile to conduct a series of tests on 12 subjects following approximately the same time routine as that used in the preceding series, but without the factor of smoking; consequently, photographs were taken of the uvula of 12 subjects who claimed to have never smoked and who did not smoke during the test, and five minutes after the first photograph a second one was taken. The results of the measure-

ments of these films, as in the previous series, may be calculated in several ways. The average response of all of the subjects showed a 10.7 per cent increase in the diameter of the blood vessels in the second photograph over the first. The average response of all of the blood vessels showed a 16.45 per cent increase in diameter. The response of an average blood vessel was 14.35 per cent increase, and the response of an average subject was 10.57 per cent increase.

Of all of the subjects used, six showed an average increase, two showed an average decrease, and three showed no change. Of all of the blood vessels measured, 21 showed an increase, three showed a decrease, and 25 showed no change. The magnitude of these changes is considerably greater than those observed in either of the two preceding series and statistical analysis of the data representing the average change of all of the blood vessels showed a highly significant difference between the two measurements.

DISCUSSION.

Measurements were lacking for the photographs of some of the subjects. These deficiencies were of two kinds: those on which no measurements were possible on any of the photos of the subject and those on which measurements were possible on only a portion of the photos of the subject. Where no measurements were possible on any of the photos, we conclude that there were no blood vessels sufficiently close to the surface to be well defined in the picture; in fact, there were many subjects who had no vessels visible to the eye. The proportion of subjects in the entire investigation on whom no measurements were possible was 32 out of 136, or 23.5 per cent.

There was a second group of subjects whose blood vessels were measurable on some frames but not on others. The explanation for this lies in the fact that these subjects had uvulas and soft palates which were sufficiently mobile so that they were out of the field of focus at the time of taking some of the pictures. The subjects in this category numbered 28,

or 26.9 per cent of the 104 subjects on whom some measurements were possible. Since in spite of the fixed distance from the camera to the subject's teeth, the above indicates the possibility of movement of the uvula and soft palate with respect to the camera lens, it seems necessary to consider the effect of this movement on the size of the image on the assumption that the movement may have been insufficient to throw the image out of focus. Of course, movement can also occur which is too rapid to be stopped by the shutter speed, but there seemed to be few, if any, examples of this.

While there are points of exact focus for every lens, there is also a range called depth of field (miscalled depth of focus) wherein the objects form superficially sharp images. The depth of this field is dependent on the focal length of the lens, the distance from the lens to the object, the diaphragm opening, and "circle of confusion," which is the size of the smallest image on which definition is needed. Since we found that the average diameters of the images of the blood vessels measured was slightly more than 0.002 inch, it seems proper to use this figure as the diameter of the circle of confusion in calculating the depth of field. From this information may be calculated the possible variations in size of the images of the blood vessels if the distance from the lens to the vessels varied over the range represented by the depth of field. The results of these calculations show that the measurable image size can vary by about $\pm$ 5.5 per cent from its size at the point of principal focus at the extremes of the field depth. This means that any measured differences observed between two consecutive photographs must be greater than about 11 per cent in order to be certain that the differences are due to actual change in size of the object and not due to change in image size because of the change in distance from the lens to the object within the zone of critical focus. In this investigation the measured changes in individual blood vessels usually greatly exceeded this "instrument error" of 11 per cent. In most instances where more than one vessel was measurable for a given subject, it was found that some of the vessels increased, some decreased and others did not change in size;

this phenomenon could not occur solely as the result of changes in distance from lens to object when the object is substantially in one plane. Furthermore, no attempt is made to draw any conclusions from the measurements of a single subject except as he becomes a unit in the calculation of averages. Hence the error introduced by movement of the object with relation to the lens can be disregarded.

In the light of the results obtained in Series UC, where a substantial difference in the size of the blood vessels without smoking was observed, the question naturally arises as to whether or not these results negate the observations made in Series UA and UB. While the exact answer to this question is not known, on the basis of the following hypotheses it appears that the results obtained with the non-smokers do not vitiate those obtained with the smokers.

1. The vasomotor control of blood vessels may be divided into two phases, the first being the phase which controls the tone of the blood vessels, and the second which alters the size of the blood vessels. It is possible that the presence of an agent affecting vasomotor control would have a tendency to make the blood vessel sizes and changes in the blood vessels more uniform than would be the case in the absence of that agent. Hence with the non-smokers the blood vessels varied in size within relatively large limits.

2. The activity of the parts under consideration may have some bearing since it has been proved by many investigators that when a part of the body is put under stress there is a response of the blood vessels generally to increase the blood supply to the part under stress. It may be that simply opening the mouth and breathing through it and the nose simultaneously may apply a stress to the mucosal surfaces of the mouth and pharynx which results in the above response of the blood vessels. In the presence of a substance which has a definite action on the blood vessels, it is possible that these changes may be more uniform and not as extensive as in the absence of such a substance; therefore, the blood vessels

observed on the non-smokers varied considerably more in size than those of the smokers.

If the data on the non-smokers are ignored and only the data in the other two series considered, one prominent feature appears; namely, that there is no consistency even with individual subjects in the effect on the diameter of the blood vessels following smoking. When the data were examined for the subjects on whom duplicate smokes were obtained it was found that the changes following the smoking of the control cigarettes were:

Test No.	% Change
61 and 86	11.1 and 5.8
56 and 102	7.1 and —4.1
63 and 111	22.2 and 0.0
26 and 110	7.3 and —7.0
59 and 109	10.0 and 0.0
34 and 73	—21.5 and 26.1

It is, therefore, evident that there is no consistent effect of smoking on the blood vessels of an individual subject and it could consequently not be expected that a consistent effect would be observed on the blood vessels of a group of subjects. In some instances, for example in Series UA, 30 of the blood vessels showed an increase in diameter, 24 showed a decrease and 51 showed no change. This could be taken to mean that in approximately 50 per cent of the cases smoking had no effect on the appearance of the blood vessels and in the balance of the cases about half of the blood vessels showed some evidences of vasodilation and the rest showed evidences of vasoconstriction.

During the course of the experiment visual observations were made on all of the subjects and in very few cases was a substantial gross change in the appearance of the tissues observed. A few of the subjects showed greatly increased reddening of the uvula and surrounding tissues.

From the practical viewpoint the importance of an investigation of this sort lies in the relative irritating effects from the smoking of the various types of cigarettes rather than slight vasoconstricting or vasodilating effects; consequently,

consideration should be given to the validity of the assumption that the effects on the blood vessels constitute indices of degrees of irritation. It seems reasonable to believe that the application of an irritant should result in a vasodilation at the point of irritation providing no other concomitant reaction occurs. Such a localized reaction would ensue from mechanical irritation or from chemicals which have no other physiological effects, although there is evidence[5,7] that generalized vasoconstriction occurs following mechanical irritation and even deep breathing.

In the case of tobacco smoke, there are many components, some of which are locally irritating and others which have systemic effects such as vasoconstriction. Thus it seems unlikely that a clear-cut evaluation of irritating properties can be made by studying the effect on the blood vessels or throats of smokers since there are always two types of agents present in the smoke having opposite effects. The result of these two opposing forces is unpredictable since it is dependent on a variable individual susceptibility and on smoking habits; consequently, as was observed in this investigation, some subjects showed an increase in blood vessel diameter and with it a probable increased redness of the area, others showed a decreased blood vessel diameter and probable attendant blanching of the area, and others showed no change. In view of these facts, it seems to us futile to attempt to evaluate the irritating qualities of tobacco smoke by such methods used here or by observing color changes in the throat unless some technique can be devised to isolate the localized irritating effects from systemic reactions.

The systemic reactions might possibly be reduced by instructing the subjects to inhale as little of the smoke as possible; however, it is practically impossible to smoke without inhaling some of the smoke, so the systemic reaction would not be eliminated. In fact, Baumberger[6] has shown that the non-inhaling smoker retains about two-thirds of the smoke, while on inhalation about 88 per cent is retained. Furthermore, Mulinos and Shulman[7] have reported that their subjects showed vasoconstriction from puffing without inhal-

ing, although not to so great a degree as from 10 deep breaths or inhaling cigarette smoke.

If the numerical data obtained in this investigation were to be considered as indicative of the relative irritating effects of different types of cigarettes, one could theoretically devise a blend of tobacco which would be apparently non-irritating according to these tests simply by increasing the proportion of vasoconstrictors, such as nicotine, to a point where all indications of irritation would be masked. On the basis of our data the blend in the "C" brand cigarettes appears to approximate such properties since the average changes observed were preponderantly zero or negative, yet it seems unlikely that the smoke from these cigarettes is really non-irritating.

In order to check the effects on light smokers and heavy smokers, the average changes following the smoking of the control cigarettes were compiled for the subjects who smoke less than five cigarettes per day and compared with similar data for the subjects who smoke 20 or more per day. It was found that the light smokers showed an average increase of 3.0 per cent and the heavy smokers showed an average increase of 4.4 per cent, but the difference was found to be not significant.

CONCLUSIONS.

As a result of the measurements of blood vessels on 544 photographs of the uvula and soft palate of 136 subjects, the following observations were made:

1. In a series of photographs of 12 subjects the measurement of the 105 measurable blood vessels showed that diethylene glycol treated cigarettes had a greater dilating effect on the blood vessels of the uvula and soft palate than either glycerine treated cigarettes or cigarettes containing no hygroscopic agent; however, the differences were so small that they were not statistically significant.

2. In a series of photographs of 112 subjects the measurement of 504 measurable blood vessels shows that there was

no difference in the effect on the diameter of the blood vessels between the smoke from the control cigarettes and that from four other leading brands taken as a group; however, it was found that the smoke from the control cigarettes appeared to have a greater dilating effect than smoke from Brands "C" and "B" and that smoke from Brand "D" had a greater dilating effect than the controls. There seemed to be little difference between the controls and Brand "E."

3. In a series of photographs of 12 subjects who claimed to have never smoked and who did not smoke during the test, measurements of 49 measurable blood vessels showed a substantial increase in the diameter of the blood vessels in the five-minute interval between the two photographs of the subject.

4. The possible correlation between the effects of smoking on blood vessel diameter and the irritating properties of the smoke is discussed and it is concluded that the conflicting effects of some of the smoke constituents invalidate methods for evaluating smoke irritation which depend on measurement of blood vessel diameter or the attendant gross color changes.

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TESTS FOR SELECTION OF HEARING AIDS.*

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I. – INTRODUCTION.

The research and clinical experiences of the past three years have yielded new approaches to the task of hearing aid selection. Many concepts which guided our thinking have been revised. For example, it is no longer possible to assume that an adequate hearing aid will be insured simply because the instrument's response curve complements the audiogram.[1] Similarly, the methodology of obtaining aided audiograms in sound fields has encountered serious practical limitations and important theoretical objections.[2]

Obviously, the final criterion of hearing aid excellence is the success with which the instrument functions in everyday situations; thus, selection procedures need to be chosen so as to yield estimates of the future usefulness promised by each hearing aid. The choice, however, must allow for the fact that in everyday situations the patient and his instrument function as a totality; furthermore, procedures must be as economical of time and equipment as possible. Most important, however, tests must be arranged and conducted so as to isolate unusual cases and give a sound basis for dealing with patients having special problems.

With the foregoing in mind, various dimensions of hearing aid performance emerge as worthy of exploration. These dimensions are:

1. Sensitivity, or effective gain;

2. Tolerance limit, or psychophysical ceiling;

3. Efficiency in background noise, or signal-to-noise ratio;

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4. Discrimination, or efficiency in distinguishing small sound differences.

The remainder of this paper discusses procedures which have been found useful in exploring these four dimensions. Specifically, the paper describes a battery of tests developed in the Acoustic Clinic at Deshon General Hospital, Butler, Pa. The battery helped reveal patients who faced special problems. It also gave estimates of the efficiency with which different hearing aids could be expected to serve each patient. All tests utilized speech as the stimulus material — both to reduce testing time and because ability to hear speech is the auditory requirement of greatest importance in everyday life.

II. — A TEST OF SENSITIVITY.

The speech reception technique offers a good method for estimating the sensitivity yielded by a hearing aid.[3]

Basically, the speech reception procedure is simple. It consists of determining the sensation level at which the patient can hear sufficiently well to respond correctly to 50 per cent of the test items. The subject's auditory acuity as measured by speech reception is the difference between the normal threshold and the sensation level at which his threshold lies. All test items consist of speech samples. They may be presented either by monitored live-voice or from phonograph records.[4] Subject responses may be either oral or written. The crux of the technique is to present appropriate speech samples through a high quality amplifying system which allows a wide range of controlled intensities to be brought to the subject.[5]

A speech reception threshold obtained through a hearing aid reveals the "residual loss for speech" which the patient experiences with that particular instrument. The sensitivity of various instruments may be compared either on the basis of the "residual losses" they yield or in terms of their relative effective gains—which are computed by taking the differences between the patient's unaided threshold and his various aided thresholds.

In order to obtain a speech reception threshold through a hearing aid, the test items must be presented in a sound field. This means using a loudspeaker in an appropriately sound-treated room. The position at which each hearing aid is tested must be carefully standardized.

The steps in a practical procedure for determining the sensitivity of hearing aids are as follows:

Step 1. The patient's unaided speech reception threshold is first determined. The primary purpose here is to obtain a point of reference against which to compare scores yielded by various instruments. To obtain the unaided threshold, the patient faces the loudspeaker with his head in the position the hearing aids will later occupy. The test is then administered.

Step 2. Sensitivity of the first hearing aid is now measured under conditions simulating the volume control adjustment which the patient would be expected to employ while listening to faint everyday speech. A good method is to ask the patient to adjust the volume control until speech striking the hearing aid at 40 db. above normal threshold is stimulating the patient in such a manner that he judges the reception most comfortable.[6] Once this setting has been made, a speech reception threshold is obtained. The "residual loss for speech" (under the conditions of tests) is the difference between normal threshold and the aided threshold thus obtained. Similarly, the effective gain for speech is the difference between the unaided threshold obtained in Step 1 and the aided threshold.

Step 3. Sensitivity at full volume is next measured by adjusting the instrument to this setting and obtaining a speech reception threshold. The "residual loss for speech" and the effective gain at full volume are computed in the manner just described.

Step 4. Steps 2 and 3 are repeated with as many instruments as the investigator wishes to explore.

The usefulness of a test of sensitivity in differentiating among hearing aids varies with the patient. Some individuals experience ample and essentially equivalent sensitivity with several hearing aids. Others receive ample sensitivity with several instruments but show differences which are great enough to constitute presumptive evidence in favor of one "fitting." Still other patients show sensitivity differentials which are sufficiently great to warrant making these differentials the primary basis for selection of a particular instrument. As a generalization — although there are obviously individual exceptions — it may be stated: *1.* that differences between instruments of 6 db. or less may ordinarily be disregarded, *2.* that hearing aids for which the "residual losses for speech" do not exceed 15 db. will have ample sensitivity for everyday situations, and *3.* that the significance of sensitivity as a criterion for selection becomes progressively greater in proportion to the degree by which "residual loss for speech" exceeds 15 db.

III. – A TEST OF TOLERANCE LIMIT.

It is easy, in conjunction with the tests of sensitivity, to estimate the strength of the sound field in which a particular hearing aid becomes intolerable for the patient.

Increasingly higher levels of speech are presented both to the unaided ear and to the hearing aids under test. Hearing aids are set at the same volume control adjustments used to explore sensitivity. The patient is asked to report the point at which he experiences some definite sensation such as pain, tickle, vibration or dizziness. The sound-field level at which such sensation occurs is taken as the tolerance limit, or psychophysical "ceiling," of the hearing aid. Care must be taken to insure that the patient is not reporting as his tolerance limit the point where the instrument becomes unpleasantly loud.

In conducting the tolerance test, it is best to use samples

of connected speech. Each sample must be sufficiently long to allow the patient to judge whether or not the sound is still tolerable. Ordinarily, three or four sentences will suffice.

Since a test of tolerance limit is somewhat disturbing to the patient, one should ordinarily increase the intensity of successive test samples by increments of 5 to 10 db. each; furthermore, it is safe to assume that if the patient has not reached the tolerance limit by the time the test sample striking the hearing aid is 80 db. above normal threshold, the "ceiling" with the hearing aid is ample for all practical purposes.

Most patients do not experience tolerance difficulties at either a "comfort level" or a full volume setting of any good instrument. A fair proportion, however, show difficulty either with the full volume setting or under both conditions. Such problems may be expected most frequently among elderly patients. When these problems occur, they encompass facts which must be weighed carefully in advising the patient. It is obvious that, whenever other factors are approximately equal, the hearing aid selected should be the one with the highest tolerance limit. If the "ceiling" is reached with all instruments at relatively low levels of input signal, the tolerance limit may justifiably be made the primary criterion for selection. Finally, the occurrence of low tolerance limits at "comfort level" settings with all hearing aids indicates that the patient faces special difficulties in hearing aid use. It is doubly imperative that he be adjusted to his hearing aid through a careful course of indoctrination and auditory training. Such training may allow his tolerance limit to be raised[7] and it can help him to avoid developing an antagonism to his instrument which will keep him from using it most effectively.

IV. – A TEST OF EFFICIENCY IN NOISE.

Since ordinary listening situations include varying degrees of background noise, it is pertinent to estimate the effectiveness with which each hearing aid performs for the patient in the presence of noise. A feasible criterion is to determine

the maximum amount of noise which can be present without destroying the ability to understand speech. A practical procedure is to determine signal-to-noise ratio.

The following steps have proved a useful method for comparing hearing aids on the basis of signal-to-noise ratio:

Step 1. The patient adjusts the volume control of the first hearing aid by the "comfort level" method. This time, speech which impinges on the hearing aid at a sensation level of 50 db. is used.[8]

Step 2. Speech samples (which may be similar to those used in obtaining speech reception thresholds) are now presented to the patient at a sensation level of 50 db. Noise at controlled intensity is simultaneously introduced into the test chamber. The sensation level at which this noise is presented is progressively increased, the patient being required at each noise level to reproduce orally several test items. The procedure is continued until a noise level is reached at which the patient can no longer understand the test items. The intensity of the noise is then dropped by slow steps until understanding returns.

Step 3. The signal-to-noise ratio is computed. The ratio is here defined as the difference between the sensation level (50 db.) at which speech is presented and the sensation level of the strongest noise with which repetition of test items is possible.[9]

Step 4. The procedure is repeated with the other hearing aids under test.

Various kinds of noise may be selected for use in establishing signal-to-noise ratio. However, the noise chosen must conform to two requirements: first, it must be acoustically broad and complex, that is, it must contain components which cover a wide frequency range; second, it must be sufficiently stable to maintain approximately the same intensity at all times.

The writer has found two noises particularly useful. One is a combination of thermal hiss and static pulses. The thermal element presents a broad background containing components at roughly equivalent intensities and covering a wide frequency range. Superimposed upon this background are sharp pulses of sound (static), which are somewhat analagous to random aspects of many everyday noises. The second useful noise is produced by generating a sawtooth wave. Acoustically, the resultant sound may be considered as consisting of a fundamental and an extended series of strong harmonic overtones. Good results have been obtained with a sawtooth wave having a base frequency of 120 c.p.s.

The numerical value of a signal-to-noise ratio depends partially upon the type of noise used and partially upon the interaction between the hearing aid and the patient's hearing loss. A person using the signal-to-noise technique must establish norms of performance in his testing situation. Once these norms are known for a reasonable sample of patients, it is possible to estimate the significance of individual scores. In general, there will be an upper limit of performance which only a few patients exceed and which the majority approach. Some patients will show poor hearing in noise.[10] As with other measures of hearing aid performance, there are patients who achieve essentially equal efficiency in noise with a number of instruments. Other patients show relatively large differences from one instrument to another. When the latter is the case, performance in background noise becomes an important criterion for hearing aid selection; furthermore, the patient who does poorly with all instruments may be expected to find his hearing aid disturbing in many everyday situations. Patients of this last type require extensive auditory training and special orientational instruction.

V. — A TEST OF DISCRIMINATION.

The ability to distinguish small differences in sound is of primary importance to the hearing aid user. It is, therefore, necessary to have a good estimate of the relative discrimination which the wearer achieves with different hearing aids.

The value of a discrimination score, of course, depends upon the adequacy of the test material. The problems involved in constructing a satisfactory discrimination test are many. Most speech tests, even though they may be called discrimination tests, are not sufficiently well constructed or thoroughly calibrated to justify their use. The most satisfactory test which has come to the writer's attention is the series of phonetically balanced word lists prepared by the Psycho-Acoustic Laboratory at Harvard University.[11] The tests are scored on a percentage basis, a word being counted wrong unless it is reproduced perfectly.

A simple procedure for estimating the discrimination allowed by different hearing aids consists of the following steps:

Step 1. Whenever possible, an unaided discrimination score is obtained by presenting the test at a level 25 db. greater than the unaided speech reception threshold.

Step 2. The first instrument is adjusted so that speech at the 40 db. sensation level is being received comfortably.

Step 3. The aided speech reception threshold is then determined.

Step 4. A discrimination test is next administered. The test is presented at a sensation level which is 25 db. above the aided threshold obtained in Step 3.

Step 5. Steps 2 through 4 are repeated with the other hearing aids being evaluated.

When such a procedure is followed, the patient may be expected to obtain approximately as good a discrimination score as is possible at any level of presentation. Significant differences between instruments may be taken as indicating differentials in the clarity of hearing which each instrument allows.

The procedure just described was used at Deshon General Hospital. It was gratifying to discover that most patients

obtained excellent discrimination scores with each of several hearing aids. One concludes that today the better hearing aids bring the majority of users high clarity in reproduction. However, some patients obtained discrimination scores which differed from one instrument to another. Whenever discrimination scores differed by 8 per cent or more, the discrepancies were generally taken as indicating sufficient distinction in instrument performance to warrant selecting the hearing aid which yielded the superior score.

A second contribution of the discrimination test was that it isolated patients for whom fine sound discriminations were difficult. On the basis of test scores, it was possible to make more meaningful recommendations regarding the special training such patients required. Furthermore, if a patient could not expect full efficiency from any instrument, he was acquainted with the difficulties he faced.

Discrimination test techniques may be adapted to secure even more detailed information; however, the procedures involved make doing so relatively impractical in the clinical selection of hearing aids. A method which has proved effective in research is to ascertain the sharpness of discrimination for different input levels of speech.[12] This method allows one to specify either the point at which a particular degree of discriminatory excellence is achieved or the range of speech intensity over which a stated degree of excellence is maintained. Such information should offer a more precise basis for choosing among instruments, although its added value can be determined definitely only by studying the relative performance of commercial hearing aids when tested on an ample sampling of patients.

VI. — A PRACTICAL TESTING SEQUENCE.

The preceding discussion has presented a brief summary of four different types of test. At Deshon General Hospital these tests were interwoven in a routine procedure which allowed three hearing aids to be explored in an hour.[13] All measures on one hearing aid were completed before the next

instrument was subjected to test. In brief, the steps were as follows:

Step 1. Three measures without a hearing aid were obtained to allow later comparison between unaided hearing and performance with the various hearing aids. The three measures were: unaided speech reception threshold, unaided tolerance limit, and unaided discrimination score.

Step 2. The volume control on the first instrument was then adjusted to the 40 db. "comfort level" and the aided threshold and the tolerance limit were measured.

Step 3. The instrument was set at full volume. The aided threshold and the tolerance limit were again determined.

Step 4. The instrument was adjusted to the 50 db. "comfort level" and two signal-to-noise ratios were obtained. One ratio was obtained in the presence of the thermal-static noise, while the sawtooth noise was used for the second ratio.

Step 5. The instrument was again adjusted to the 40 db. "comfort level." The aided threshold was again obtained. Comparison of this threshold with the earlier threshold under similar conditions allowed a more precise estimate of the sensitivity yielded by the instrument at a "use" setting. A 50 word discrimination test was then administered. Care was taken to present the discrimination test at a level 25 db. higher than the threshold just obtained.

Step 6. Steps 2 through 5 were repeated with the second hearing aid and, eventually, with the third instrument.

The foregoing procedure yielded seven scores on each instrument. These seven scores covered the four dimensions: sensitivity, tolerance, efficiency in noise and sound discrimi-

nation. The composite of results made possible the selection of a hearing aid better suited to the patient's individual needs. While it is true that many patients obtained equivalent performance with different hearing aids on one or more of the test items, every item differentiated performance in a sizable proportion of cases. Selection was made sometimes in terms of one criterion, sometimes in terms of another criterion, and sometimes in terms of several criteria. With patients for whom performance was equivalent on several instruments, selection could be based on auxiliary factors of convenience, weight and esthetic preferences.

Another value of the procedures was that it picked out and called sharp attention to the special problems of hearing aid use which some patients have. From tests results, it was clear in certain instances that one instrument possessed sharp superiority. It was clear in other instances that further study of the case was required. A complete reassessment of hearing aid fittings was at times necessary. Finally, special problems of hearing aid use were frequently highlighted. It was thus possible through interviews and emphasis in auditory training to adjust the patient to his instrument much more intelligently.

VII. – GENERAL COMMENTS.

The tests described above are applicable to widely varied conditions for selection of hearing aids. They can be used as the final step in a detailed series of screening procedures. By contrast, they can be employed in situations where only a limited time is available with each patient.

When limited time is available the methodologies are particularly useful, although certain special problems appear.[14] Not only do the tests yield a relatively quantitative description of performance but they also give a series of measures which point sharply to any special problems of hearing aid use which the patient may have. A skilled examiner can interpret the patient's scores so as to emphasize special needs. *It is thus possible to serve not only the function of selecting*

a superior instrument for the patient, but also to do so in such a manner as to protect the person with unusual requirements by defining his problem more fully. However, there are patients with whom the procedures cannot be used immediately or in their entirety. Liberal adaptation of methodology is sometimes necessary. Children — particularly youngsters with hearing losses of long standing — often lack the language ability and general understanding required for the test procedures. Adults who are unfamiliar with hearing aids sometimes find phases of the test difficult unless there is previous indoctrination. Foreign born patients are likely to fall in this category. Elderly persons constitute the group most frequently experiencing difficulty. Initially, such elderly persons often received relatively little help from a hearing aid. The sensitivity (effective gain) they achieve is poor and they frequently evidence low tolerance limits. Fortunately, adaptations of the procedures described in this paper can help define the nature and severity of such special problems. Clear recognition of the special problems points to the particular training needs which are required to make the patient a more effective hearing aid user.

VIII. – CONCLUSION.

The field of hearing aids is in rapid flux. Substantial changes in instrument design and performance are occurring. Our understanding of patient requirements and of the important criteria for instrument selection are becoming more definite. We may expect the confusion which has clouded the field to dissipate steadily.

While it is impossible to predict the details of future developments, six guiding principles can be stated.

1. The patient must be protected by methods of hearing aid selection which insure him of sensitivity, tolerance, performance in noise and discrimination which are as adequate as his case allows.

2. Regardless of what we discover about the requirements of the majority of patients, individuals with unusual

problems and special needs will constitute a persistent challenge.

3. The clinical routines one chooses must include tests which will isolate and define the problems of the patient having unusual needs.

4. It is desirable to employ clinical routines which yield quantitative scores. Such scores allow an individual's results to be compared with those obtained by other hard-of-hearing persons.

5. Clinical routines must — within the limits required by the foregoing principles — be as simple as possible.

6. Every hearing aid user can benefit from auditory training. The details of the training program may range from simple to complex. They must be selected to meet the individual's needs.

In conclusion, the tests described in this paper were designed to serve the foregoing principles, and have proved clinically effective in differentiating among hearing aids and in defining the special problems facing unusual cases.

REFERENCES.

1. For one thing, today's hearing aids offer a relatively limited range of frequency response characteristics. Most audiograms can be only imperfectly complemented. More fundamental, however, has been the amassing of clinical and research evidence favoring the view that the majority of hearing loss cases obtain superior results with instruments having either flat responses or slight rises in sensitivity for higher frequencies. See Davis, H., and Ross, D. A.: Selection of Hearing Aids. Cambridge, Mass.: Harvard Univ., 1945. See also, Davis, et al.: The Selection of Hearing Aids. THE LARYNGOSCOPE, 56:85-115 and 135-163, 1946.

2. Op. cit., 15-17.

3. Hughson and Thompson routinely employed the method in hearing aid selection. They used two operators. Subsequent improvement allows a single person to administer the test. See: Hughson, W., and Thompson, E.: The Hearing Aid from the Patient's Point of View. *Arch. Otolaryngol.*, 38:252-260, 1943; Carhart, R.: The Selection of Hearing Aids. *Arch. Otolaryngol.*, 44:1-18, 1946.

4. Several groups of test material are available.
Hughson and Thompson used the Bell Telephone Intelligibility Lists, which are presented by live-voice.
Recently, the Psycho-Acoustic Laboratory at Harvard University pre-

pared lists of spondaic words. These word lists are thoroughly calibrated and may be presented by either live-voice or phonograph record. These word lists, as well as a companion series of recorded questions, will prove highly useful for both clinical work and research.

Several lists of sentences have been prepared to meet the needs of patients having special requirements. For example, a series covering common experiences in the Army was prepared at Deshon General Hospital for use with servicemen of limited intelligence or language ability. Another list especially for children has been developed at Northwestern University. Currently, most such lists must be presented by live-voice.

5. CARHART, R.: Monitored Live-Voice as a Test of Auditory Acuity. *Jour. Acous. Soc. Am.*, 17:339-349, 1946.

6. If one is going to measure sensitivity at a "use" setting, a method must be employed which yields equivalent settings with different hearing aids. The problem of finding a practical way for accomplishing this requirement is complex. Without attempting full analysis here, it may be stated that the "comfort level" procedure described above has proved clinically effective. Among other things, it has been shown that patients who have received indoctrination in the use of hearing aids obtain repeat thresholds on independent "comfort level" settings which are within the limits of accuracy ordinarily accepted in clinical audiometry. It is the writer's opinion that the "comfort level" method is a feasible clinical procedure for obtaining reasonable equivalence in adjusting the volume controls on different instruments. Extra precision of results is possible while evaluating hearing aids if two independent thresholds at the same "comfort level" are obtained with each instrument. See Carhart, R.: Volume Control Adjustment in Hearing Aid Selection. THE LARYNGOSCOPE, 56:510-526, 1946.

7. DAVIS, H., and ROSS, D.: Op. cit., 21-22.

8. The reason for selecting this level is that in the noisy situations of life speech tends to be more intense than it is in the quiet situations. Setting the instrument for a sensation level of 50 db. represents a more reasonable simulation of life conditions than setting the instrument for 40 db. speech.

9. The signal-to-noise ratio is often defined, in audiological research, as the difference between the sound pressure levels of the speech and the noise. Such usage has the advantage of describing the two sounds in terms of a common reference point on the scale of physical intensities. From the clinical standpoint, however, such usage has the disadvantage of demanding that test equipment be calibrated in terms of sound pressure level. Calibration on this basis is often difficult to accomplish under clinical conditions of sound field measurement. The problem is further complicated by the acoustic complexity of the noise and the acoustic fluctuations which characterize speech. It is therefore much more practical in the evaluation of hearing aids to describe each type of sound in terms of its sensation level and to take the difference between sensation levels as the signal-to-noise ratio. When this is done, it is merely necessary to calibrate by establishing the threshold for each type of sound on ears known to be normal. The normal threshold for the noise will not lie at the same sound pressure level as the normal threshold for speech, but this discrepancy can be disregarded in making clinical judgments as long as the same testing conditions are preserved.

10. The occurrence of extremely poor hearing in noise calls for particularly careful interpretation. When no good reason for the difficulty

can be found, such a result raises the presumption that the hearing loss may be at least in part of psychogenic origin.

11. This test is known as the PB-50 Test. See *Articulation Testing Methods II*, OSRD Report 3802, November, 1944.

12. Davis, H., and Ross, D. A.: Op. cit., 23-24.

13. The tests described constitute what was termed the Hearing Aid Evaluation. The Evaluation was the final stage in a series of screening and selection procedures. Only those instruments were subjected to evaluation which proved in earlier screening stages to be most promising for the patient. For description of the other procedures, see Carhart, R.: The Selection of Hearing Aids.

14. The instruments to be given final test must here be selected arbitrarily by an expert on the basis of medical examination, case history, audiometric findings, and speech reception data.

MISSISSIPPI VALLEY MEDICAL SOCIETY 1947 ESSAY CONTEST.

The Seventh Annual Essay Contest of the Mississippi Valley Medical Society will be held in 1947. The Society will offer a cash prize of $100.00, a gold medal and a certificate of award for the best unpublished essay on any subject of general medical interest (including medical economics and education) and practical value to the general practitioner of medicine. Certificates of merit may also be granted to the physicians whose essays are rated second and third best. Contestants must be members of the American Medical Association who are residents of the United States. The winner will be invited to present his contribution before the Twelfth Annual Meeting of the Mississippi Valley Medical Society to be held at Burlington, Iowa, Oct. 1, 2, 3, 1947, the Society reserving the exclusive right to first publish the essay in its official publication—the *Mississippi Valley Medical Journal* (Incorporating the *Radiologic Review*). All contributions shall be typewritten in English in manuscript form, submitted in five copies, not to exceed 5,000 words, and must be received not later than May 1, 1947. The winning essays in the 1946 contest appear in the January, 1947, issue of the *Mississippi Valley Medical Journal* (Quincy, Ill.).

Further details may be secured from Harold Swanberg, M.D., Secretary, Mississippi Valley Medical Society, 209-224 W. C. U. Building, Quincy, Ill.

VOICE CHANGES IN MALE ADOLESCENTS.

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Recent articles by Fairbanks,* Pedrey† and Curry‡ have described certain pitch aspects of voice change in male adolescents. A growing interest in the whole development of the adolescent voice has been indicated by investigators in the fields of speech, music, psychology, anthropology and medicine. It is the purpose of this article to summarize the work of these investigators by outlining answers for the four following questions:

1. Perceptually, how is voice change characterized?
2. At what ages may this change be expected to commence?
3. How can this change be explained on an anatomical, physiological or acoustical basis?
4. What theories for this particular time of change of voice have been advanced?

1. Perceptually, how is voice change characterized?

Voice change has long been evident, especially to teachers of speech and music, because of distinctive perceptual deviations from the normal voice pattern. These deviations have been reported by Calver, Henley, Hipsher, Jerome, McCarthy, Negus, and Seth and Guthrie. These observers were primarily impressed by the loss of vocal control and general instability of the voice undergoing change. Jerome has presented a most picturesque description of this deviation:

"Change of voice may be technically described as a normal

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*Fairbanks, Grant: Recent Experimental Investigations of Vocal Pitch in Speech. Jour. Acous. Soc. Am., 457-466, 1940.

†Pedrey, Charles P.: A Study of Voice Change in Boys Between the Ages of 11 and 16. Sp. Monog., XII:30-36, 1945.

‡Curry, E. Thayer: The Pitch Characteristics of the Adolescent Male Voice. Sp. Monog., VII:48-62, 1940.

loss of control of voice, occupying an undetermined length of time in the preadolescent stage or during some period of adolescence. The loss of control is usually evidenced by instantaneous, unpredicted and involuntary changes of pitch, the reasons for which are not readily apparent. Occasional concomitants of this loss of control of the voice are double resonance, restricted range of pitch and the appearance of a tremelo."*

This tendency toward a loss of control of the voice in most boys is further substantiated by Seth and Guthrie: " . . . so that control of the voice becomes uncertain. In boys the voice may become hoarse and raucous with a tendency to 'break' into falsetto notes higher than those which were normal before the change began."†

Pedrey had defined the voice "break" as follows: "A voice break is here defined to mean a sudden and uncontrollable rise or fall from the characteristic pitch of the individual, a rise or fall which is definite enough to be heard by a listener."‡

Some music teachers have noted the general range of tones within which this "break may be expected to occur. Calver, writing for music teachers, has suggested the following tests to determine if changes are taking place: "If a boy be asked to sing up the scale of, say, C, it will be noticed that, somewhere about G_1 or A_1 he makes a natural change of tone. The first note, C, is always more or less robust; but, when he reaches G or A, his voice becomes thinner and, in the case of an untrained boy, decidedly weaker, unless it is obviously forced."§

Investigators in this field are rather agreed on the extent of lowering of the voice. Birge, Seth and Guthrie, Nadoleczny, Dawson, Cain and Curry all report that the voice is lowered an octave over the period of the change. Typical of these views is Nadoleczny: "The voice of boys gradually sinks by an octave, that of girls only by a third. The speak-

*Jerome, Eldon K.: Change of Voice in Male Adolescents. Quar. Jour. of Speech, XXIII:648-653, 1937.

†Seth and Guthrie: Speech in Childhood, pp. 200-201, London, 1935.

‡Pedrey, C. P.: Op. cit., p. 32.

§Calver, F. L.: Training Boys' Voices. Etude, 49:54, 1931.

ing voice of boys suffers a corresponding change, going down to *d* or *e* in A."¶

Seth and Guthrie suggest further: "In boys the lower limit falls a whole octave and the upper limit about a sixth."** The recent investigations of Fairbanks and Curry have indicated the extent of the voice "break" to be one octave. Curry reported the findings of these two workers: "The mean, and likewise the mode, extent of breaks, both upward and downward, and for both 10- and 14-year-old groups, was found to approximate six tones, *i.e.*, one octave."*

McCarthy, Seth, and Guthrie and Mellalieu have listed symptoms which appear to precede the actual change of pitch. Mellalieu summarizes these predictions quite clearly:

"What we hear at the changing period is a change of quality and pitch. . . . The symptoms of the changing period are many. From the vocal point of view the commonest signs are:

1. The speaking voice becomes husky and lacking in control. The singing may be affected in the same way.
2. Sometimes the lower portion becomes weak and uncertain, but the upper portion remains unaltered for a time.
3. The speaking voice begins to get much heavier in tone, the light, boyish quality gradually disappearing.
4. The higher notes disappear or become difficult (notes above E flat, fourth space, treble clef), followed later by notes above B flat, but the lower portion in most cases remains and even improves in tone quality."†

McCarthy also suggests something most interesting and to which no other reference has been found: "Just before the huskiness which precedes the actual break, the boy's voice

¶Nadoleczny, Max: Speech Disorders in Childhood. Dis. Child., p. 465, Lippincott, 1914.
**Seth and Guthrie: Op. cit., p. 200.
*Curry, E. Thayer: Op. cit., p. 61.
†Mellalieu, W. N.: The Boy's Changing Voice, p. 11, London, 1935.

frequently takes on a sort of metallic ringing clearness which is very pleasing if the tone is not forced."‡

Twenty writers have contributed notes on the perceptual characteristics of voice change. Generally, the voice is described as husky before the onset of change, unstable during change, and an octave lower in pitch after change. This description of voice change leads to the second pertinent question—*When* can this change be expected to occur?

2. At what ages may this change be expected to commence?

A German, Paulsen, working about 1887, reported that 50 per cent of boys began to experience voice change at age 13; by age 14, 70 per cent were in a process of change; by age 15, 80 per cent were undergoing voice change. He further reported that 6 per cent had completed this change by age 14; 3 per cent still had the prechange voice by age 19.*

The study of Pedrey† indicates considerably different results from that of Paulsen. Of Pedrey's subjects, 87.9 per cent had begun the change at age 13 (Paulsen, 50 per cent). Further, 20.2 per cent had completed this change by age 14 (Paulsen, 6 per cent). Pedrey indicated that by age 15 only one-half of 1 per cent still had not experienced change of voice, whereas Paulsen reported that by age 19, 3 per cent still had the boy's voice.

The general literature evinces much indecision regarding any more precise determination for the time of this change in individuals. This uncertainty is particularly evidenced by Howard:

"This break of the voice, as it is called, occurs at about the age of 15 years in this climate, but often a year or two earlier, and not infrequently a year or two later. The growth of the larynx goes on, with greater or less rapidity, varying in

‡McCarthy, C. V.: The Teaching of Vocal Music to Junior High School Students. M.A. Thesis, State University of Iowa, pp. 4-5, 1937.

*Paulsen, E.: Untersuch ungen uber die Tonhohe der Sprache. Pflug, Arch., 74:570-576, 1899.

†Pedrey, C. P.: Op cit., p. 33.

different individuals, for from six months to two or three years, until it attains its final size.‡

The work of Jerome provided data which evolved in conjunction with certain anthropometric measures:

3. Boys undergoing change of voice who are of the younger chronological ages, are, in general, older skeletally than mentally or chronologically.

4. Boys undergoing change of voice who are of the older chronological ages are, in general, younger skeletally than they are mentally or chronologically.

5. Skeletal age gives a more homogeneous grouping of boys undergoing change of voice than does mental or chronological age.

6. Skeletal age would seem to be more closely correlated with change of voice than the other criteria of age used.*

To summarize this section on the temporal aspects of change of the adolescent voice, it would seem that even the objective studies of Jerome and Pedrey would indicate that there is no narrow range during which the voice change may be expected to take place. The very large range in chronological age is doubtless due to the wide range of skeletal ages which are to be found in a given group all of the same chronological age. The best evidence from Curry and Pedrey would seem to indicate that the mean age at which this change takes place is near 14 years on a chronological age scale.

3. How can this change be explained on an anatomical, physiological or acoustical basis?

The anatomical source for vocal vibrations is, of course, the larynx. The variations in growth rate of the larynx have been suggested as responsible for the voice break in the adolescent boy. The nature of this long-time growth has been pointed out by Howard:

‡Howard, F. E.: The Child Voice in Singing, p. 22, 1898.
*Jerome, E. K.: Op. cit., p. 301.

"The voices of boys and girls, prior to the age of puberty, are alike. The growth of the larynx, which is in each quite rapid up to the age of six years, then, according to all authorities with which the writer is conversant, ceases, and the vocal bands neither lengthen nor thicken, to any appreciable extent, before the time of change of voice, which occurs at the age of puberty."†

The typical view is elaborated further by McCarthy:

"While this uneven development is taking place in the large bones and muscles of the body, there is often an eccentric development taking place in the muscles of the larynx. The box, made of cartilage, may develop more rapidly than the muscles controlling the vocal cords, or the vocal cords or bands may grow so unevenly that the muscles that control them cannot direct the delicate movements necessary to voice production. This latter difficulty is probably responsible for the so-called 'break' or change in the boy's voice";‡ however, the one leading medical authority in this field, Negus, does not present an identical point of view:

"At puberty a great change in length and mass of vocal cords takes place in the male, and his voice drops to a noticeable degree. At six and a half years a considerable increase in size has taken place; the vocal cord is long and occupies well over one-half of the diameter of the glottis, while the vocal process does not extend so far into the margins of the membranous glottis. Little change in size takes place from this age until puberty, when rapid growth changes occur in the male. The vocal cord is then found to be longer and thicker, occupying about two-thirds of the margins of the glottis.

"The reasons for breaking of the voice depend on the sudden enlargement of the larynx, and particularly on lack of proper control of the more massive muscles."*

†Howard, F. E.: Op. cit., p. 19.
‡McCarthy, C. V.: Op. cit., p. 3.
*Negus, V. E.: Op. cit., p. 431.

Seth and Guthrie present a viewpoint in accord with standard anatomical materials:

"About the age of 14 years in boys, and 12 years in girls, the voice undergoes remarkable changes. The larynx then grows rapidly, so that the vocal cords become, in boys, 1 cm. longer, in girls, 3 to 4 mm. longer, and the voice alters accordingly—in boys, the lower limit falls a whole octave and the upper limit about a sixth."†

Howard gives other measurements on the size changes in the larynx:

"In boys, the larynx doubles in size, and the vocal bands increase in the proportion of 5 to 10 in length. This great gain in the elngth of the vocal cords is due to the lateral development of the larynx, for the male larynx, in its entirety, increases more in depth than in height; the result is a drop of an octave in the average boy's voice, the longer bands producing lower tones. The change in size in the female larynx is in the proportion of 5 to 7."‡

One older writer, Dawson, regards the change of voice as indeed a serious matter:

"The break means that the voice has really collapsed; has become a mass of debris—broken down, ruined, destroyed, for all but the most ordinary purposes."*

The rather pessimistic view presented by Dawson seems somewhat extreme.

A somewhat broader view on the significance of voice change as related to physical growth is presented by Jerome:

"The most important one of the findings above seems to be that the age range in months for a group of normal boys selected as unquestionably being within the period of change of voice is found to be more restricted when measured according to skeletal age than when measured according to chrono-

†Seth and Guthrie: Op. cit., p. 200.
‡Howard, F. E.: Op. cit., p. 23.
*Dawson, J. J.: Op. cit., p. 12.

logical or mental age. In other words, this might indicate that change of voice is definitely a growth phenomenon and, therefore, may vary to a large extent with either chronological or mental age, but will have to be bound up rather closely with a definite range of skeletal age. This result, of course, should be weighed in the light of possibility that skeletal age is always a more restricted measure than either chronogical or mental age, regardless of selection of the group measured."†

Pedrey observed pubic development and noted the stage of voice development. He summarized his findings:

"The onset of greatest voice change depends somewhat on public development. Almost 23 per cent of all the prepubescent boys observed still had childish voices, while slightly less than 2 per cent of them had changed voices. But 75.5 per cent of the prepubescent boys used in this study were in the period of voice change.

"At the pubescent level, 89 per cent of the boys were in the process of voice change, while slightly more than 5 per cent still had childish voices and an equal per cent had mature voices."‡

Curry, in his studies, found many more breaks than did Pedrey. This variance may be attributed to the differences in experimental method. Curry used wave-to-wave analysis of adolescent speech; Pedrey observed the boys speaking in the classroom, without recourse to recording techniques.

In summary for this section on the anatomical correlatives of the change of voice, it can be said that a definite change of size in the larynx is taking place *about* the time of change of voice. The objective studies of Pedrey and Jerome have indicated that the phenomenon of voice change is connected to both pubic development and a general physical growth pattern which would be indicated by skeletal measurements.

4. What theories for this particular time of change of voice have been advanced?

The report of this one study is included because of its

†Jerome, E. K.: Op. cit., p. 302.
‡Pedrey, C. P.: Op. cit., p. 35.

interesting speculative factors. It is well to remember that the theory presented here is that given by a widely recognized medical authority on the development, anatomy and peculiarities of the larynx. He has presented the theory out of his own research background—since the theory appears for the first time as a result of his studies.

" 'We may conclude that the greater size, strength, courage, pugnacity, and even energy of Man, in comparison with the same qualities in Women, were acquired during primeval times, and have subsequently been augmented, chiefly through the contests of rival males for the possession of the females' (Darwin). As this quotation indicates, sexual factors play a great part in the differences shown by the adult male, and changes taking place in the larynx at the time of puberty may be explained in this way.

"The increase in strength necessitated by the coming struggle for the enticement, capture and retention of a mate cause the sexually-mature male to require a greater air supply to his lungs; for this purpose the air-way through the larynx must be increased in size.

"If the vocal cords remained of the same size as in the adolescent, the inlet valve mechanism would be inefficient, as air could not be prevented efficiently from entering the lungs. Therefore, the vocal cords elongate, and the change takes place rather suddenly at the time of puberty; measurements have been given above. Because of the sudden transition, control of the glottis as a vocal organ is temporarily lost, and the voice is liable to crack, until such time as it has completely broken, and is once more under control in a lower key than before."*

It must be remembered when considering this theory of Negus, that the anatomists tell us that the use of the larynx as an organ of speech is purely a usurped and overlaid function. Viewing the larynx as primarily an organ for control of the air stream to the lungs, the theory becomes somewhat more tenable.

*Negus, V. E.: Op. cit., pp. 255-256.

SUMMARY.

In summarizing the investigations on change of voice in male adolescents, the following conclusions seem justified:

First: Perceptually, voice change is characterized by:

1. Loss of control of voice and vocal instability.
2. "Breaks," which are one octave in extent.
3. Certain symptoms, including huskiness, which precede the actual change of pitch.

Second: The mean age at which these changes take place is approximately 14 years on a chronological age scale.

Third: Certain enlargements take place within the larynx approximately at the time of change of voice.

Fourth: Objective studies have related change of voice to pubic development and certain anthropometric skeletal measures.

Fifth: A rather extended bibliography, not previously published, is included as an aid for the further work indicated in the field of voice change.

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